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CHRONIC MERCURY POISONING

BY

MONAMY BUCKELL, DONALD HUNTER, REGINALD MILTON
and KENNETH M. A. PERRY

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The most prominent symptoms of chronic mercury poisoning are erethism, tremor and stomatitis. The psychic disturbance known as erethism is the first of the symptoms to develop; it is a peculiar form of timidity most evident in the presence of strangers. The name is derived from the Greek word ' red ' and is attributed to the blushing embarrassment of the sufferer. John Pearson of London (1758–1826) first gave the symptom complex the name erethism (Almkvist, 1929). The man affected is easily upset and embarrassed, loses all joy in life and lives in constant fear of being dismissed from his job. He has a sense of timidity and may lose self control before visitors. Thus, if one stops to watch such a man in a factory, he will sometimes throw down his tools and turn in anger on the intruder, saying he cannot work if watched. Occasionally a man is obliged to give up work because he can no longer take orders without losing his temper or, if he is a foreman, because he has no patience with men under him. Drowsiness, depression, loss of memory and insomnia may occur, but hallucinations, delusions and mania are rare.

The most characteristic symptom, though it is seldom the first to appear, is mercurial tremor. It is neither as fine nor as regular as that of hyperthyroidism. It may be interrupted every few minutes by coarse jerky movements. It usually begins in the fingers, but the eyelids, lips and tongue are affected early. As it progresses it passes to the arms and legs, so that it becomes very difficult for a man to walk about the workshop, and he may have to be guided to his bench. At this stage the condition is so obvious that it is known to the layman as ' hatter's shakes.' The tremor often passes away if the patient gives up his work before it has reached a serious stage; if this is delayed the tremor may persist for 15 or 20 years. Alcoholism favours its development, and it is claimed that no total abstainer has ever suffered from tremor in severe form.

Stomatitis manifests itself by salivation and tenderness of the gums. The gums are swollen and bleed readily and, rarely, a mercurial line is seen on the gums; it usually resembles the blue line of lead poisoning, though sometimes it is dark brown. Neal and his colleagues (1941) report that dermographia, excessive perspiration, pallor and an abnormal readiness to blush are manifestations of chronic mercurialism, while Adler (1891), Jacobsohn (1920) and Tylecote (1912) have reported increased deep tendon reflexes as part of the clinical picture.

These symptoms of chronic mercury poisoning must be clearly differentiated from those of acute mercurial poisoning, which rarely arises in industry and usually results from the use of drugs such as mercury bichloride, taken in excess, either by accident or with suicidal intent. Diarrhoea is usually the first symptom: the stools, faeculent at first, soon become watery, profuse and blood-stained. They often contain pieces of mucous membrane and have a disagreeable odour. Marked tenesmus and severe pain in the lower abdomen are usually present. In some cases vomiting occurs even when the poison has not been taken by mouth. Renal symptoms are also common; there is a marked reduction in the output of urine, which may lead to complete anuria. The urine contains red and white blood cells as well as albumin and epithelial and granular casts. Headache and somnolence follow and death from uraemia usually occurs within several days.

As to poisoning by organic mercury compounds, Evans and Perry (1943) reported immediate death following the intravenous administration of mercurial diuretics in six patients, and many more cases have been reported in the United States. Hunter, Bomford and Russell (1940) drew attention to the extremely different nature of poisoning with methyl mercury compounds. Salivation, stomatitis and erethism were absent, and the nervous system alone is involved. The symptoms were severe generalized ataxia, dysarthria, and gross constriction of the visual fields, while memory and intelligence were unaffected. There was an intense and widespread degeneration of certain sensory paths of the nervous system, the peripheral nerves and posterior spinal roots being affected first; the posterior columns and the granular layer of the middle lobe of the cerebellum later.

Mercury fulminate is handled in explosives factories where detonators and percussion caps are made. The substance readily gives rise to dermatitis. Generally the uncovered parts of the body are attacked by an erythema, accompanied by intense

itching, swelling and oedema, particularly on the face, eyelids, neck, behind the ears and on the forearms. Erythematous papules break out on the inflamed areas and may become vesicles, bullae and pustules. A pustular folliculitis often develops on the hairy parts of the skin. A grain of fulminate may lodge in a crack or abrasion of the skin and act as a corrosive, causing small painful necrotic lesions, especially in the tips of the fingers (' powder holes ') which last about fourteen days (Macleod, 1916). If fulminate attacks the knuckles or roots of the nails, ulceration may penetrate to the joint and bone. Exceptionally, the whole body is affected. Recovery takes place in one or two weeks, and is accompanied by desquamation.

History of Industrial Mercurialism

Quicksilver has been mined in Spain since Roman times, and we know that mercurialism was recognized even then, for Pliny mentions it in writing of the diseases of slaves. Gabrule Fallopius (1523–1562) in his treatise ' De metallis et fossilibus ' noted that the workers in quicksilver mines suffered from mercury poisoning, and that the majority of miners remain at this work for barely three years (Rosen, 1943). Andre Mattioli of Siena, a contemporary of Fallopius, observed chronic mercurialism among the miners of quicksilver at Idria, in Carncola. Pister van Foreest (1522–1597) of Delft, also made similar observations. In 1665 tremors affecting the hands of workers in cinnabar mines in Italy led to the shortening of the working day to six hours; this is said to be the first legislative measure of industrial hygiene known to history (Hamilton, 1922). Several interesting observations relating to occupational mercurialism among miners were made during the eighteenth century. In 1719 Bernard de Lussein presented a memoir to the Academy of Sciences in which he reported the condition of the workers in the Almaden mercury mines in Spain. Both free and slave labour were employed there, and both groups of workers suffered from mercury poisoning. Nevertheless, the free miners kept themselves clean, and the only evidence of mercurialism which was exhibited was slight tremor. The slaves, on the other hand, imprisoned in their unclean quarters, and without any adequate means of washing, were afflicted with swellings of the parotid glands, stomatitis, salivation and pustular rashes. Giovanni Scopoli described mercury poisoning with the characteristic trembling among miners around Alto Isonzo. In 1804 there was a fire in a quicksilver mine in Austria and mercury vapour escaped into the air and spread over the countryside; 900 persons in the neighbourhood had mercurial tremor and many cows suffered from salivation, cachexia and abortion. In 1810 a British ship had some mercury containers broken in the hold, as a result of which all the birds and cattle on board died, and 200 persons in the ship developed symptoms of mercury poisoning, and three of them died. In 1861 Kussmaul painted a vivid picture of industrial mercurialism amongst the mirror makers of Nuremberg in the days when the method of backing mirrors with quicksilver was still used. Chronic mercurialism in the felt hat industry was first described in St. Petersburg in 1829 (Hamilton, 1922) and later reported in New Jersey by Freeman in 1860 and Dennis (1878) and in England accounts were given from Guy's Hospital in 1864 by A. G. Taylor, and in 1901 by J. G. Taylor. Reports of mercury poisoning occurring in laboratory workers using physical apparatus have recently been published (Christensen, Krogh, and Nielsen 1937; McCarroll, 1939).

Industries with a Risk of Mercurialism

Occupations giving rise to the risks of exposure to metallic mercury and its inorganic salts include mercury mining, recovery of the metal from ore, separation of gold and silver from their ores by means of an amalgam with mercury, manufacture of some types of electric meters, electric lamps and radio valves, water gilding in which an amalgam of gold or silver is applied to the object concerned and the mercury volatilized by heat, manufacture of surgical dressings containing mercury salts, bronzing of field glasses and photo-engraving, the felting of fur and the manufacture of hard felt hats, the manufacture of tungsten-molybdenum rod and wire (Lewis, 1945), the manufacture of barometers and thermometers, and in the chemical industry, particularly in the preparation of pharmaceutical compounds of mercury. According to Bloomfield and his colleagues there were 32,855 persons exposed to a mercury hazard in the United States in 1940, the principal danger being in the felt hat industry. It is from this that the phrase ' as mad as a hatter ' is derived. The fine hairs which form the fur of rabbits, hares, muskrats, and beavers are smooth, resilient and straight. Treatment with some chemical substance, and, until recently, an acid solution of mercuric nitrate, was mostly used to make them limp, twisted and rough, thus greatly aiding the felting process. Matting felt means matting fine hairs together, shrinking and pressing them till the mat is firm and hard.

Neal and his colleagues (1941) examined 534 hatters employed in five representative felt hat factories and showed that 59 had signs of chronic mercurialism. Four of the twenty-one men engaged in mixing and blowing, eight of the 34 coners, 6 of the 29 hardeners, and 33 of the 179 starters, wetters down, and sizers were so diagnosed. Mixers and blowers were exposed to 5μg. of mercury per 10 cu. m. of air, hardeners to 2·7, and starters, wetters down, and sizers to 2·1 μg. In any range of exposure above 100 μg. mercury per cu. m. the incidence of mercurialism increased with increasing duration of employment. No cases were found among hatters exposed to less than 100 μg. per cu. m. as measured by the Nordlander instrument. Workers with raised systolic blood pressure and albuminuria tended to excrete less mercury in their urine than similarly exposed workers who were normal in these respects. Patients with mercurialism were found to excrete

slightly less mercury in their urine than similarly exposed but non-affected workers. Substantial percentages of urine samples from workers with the heaviest mercury exposure were found to be free from mercury; but this finding was not confirmed in investigations subsequently carried out by Storlazzi and Elkins (1941).

Alice Hamilton (1943) stresses the fact that the Spanish quicksilver mines are dangerous because they contain pure mercury; whereas in California, Nevada, Texas and Oregon they mostly contain red cinnabar—mercury sulphide. However, in some in Sonoma County the quicksilver runs free; these are so dangerous that men became poisoned within two weeks and they have had to close down. From cinnabar a temperature of 400° C. releases the metal. It is therefore dangerous for workers to handle tobacco in such work, as the heat freely volatilizes the mercury. The men are careful not to take clothes home with them.

Mercury poisoning in industry has never been fully investigated. The factors responsible are first the difficulty of being sure that the early symptoms are caused by mercury, and secondly that of finding a method capable of identifying, differentiating and estimating mercury in micro amounts. It is impossible to distinguish early erethism from shyness or anxiety neurosis; similarly it is difficult to distinguish early mercurial tremor from that of hyperthyroidism or anxiety neurosis, especially when all the workers already know that tremor is the cardinal sign of mercury poisoning. Difficulties arise in differentiating mercurial stomatitis from pyorrhoea, which is so common in the general population, and from Vincent's stomatitis, outbreaks of which have been not uncommon in the general population during the last five years. McGeorge (1935) discussed the difficulty of this diagnosis and suggested microscopical examination of the floor of the mouth would help to differentiate, and that in chronic mercurial stomatitis there was a characteristic odour. This was not observed in the series of patients to be described.

The most intensive work on the chemical side has been carried out by Stock (1931, 1943) in Berlin, who devised a method for the estimation of traces of mercury in the air and of biological materials. It consists of bringing the mercury into solution in the form of mercuric chloride, separating the metal electrolytically on a copper wire, distilling the mercury from the wire to form metallic droplets, which are then measured with the aid of a special microscope. He claimed to be able to detect quantities of mercury as low as 0·001 μg., but obviously such a method would demand considerable experience if it is to give reliable results. He claimed that mercury was present in a wide variety of materials and that the normal human intake in foodstuffs amounted to 0·1–1 μg. per day, and that about the same amount is excreted mainly in the urine, while the normal content of the blood is about 0·1 μg. per 100 g. Teeth with amalgam fillings increased the urinary excretion from 20 to 30 μg. daily. Mercury accumulated mainly in the kidneys, but also to a minor extent in the hair, finger-nails and muscles. There is a considerable accumulation in the endocrine glands, particularly the pituitary body, and Stock considered that this explained the nervous symptoms of chronic mercury poisoning. Chemists, dentists and industrial workers, he claimed, were prone to mild chronic mercurialism, and excreted several micrograms of the metal in their urine, while the blood mercury rapidly increased after exposure. Much work was carried out on laboratory workers, and the presence of globules of mercury in floors or behind benches was sufficient to raise the air concentration to levels likely to cause chronic mercurialism. Stock stated that much larger quantities of mercury could be taken by mouth than when breathed, before symptoms became manifest.

Present Investigation

In an attempt, therefore, to throw light on the problem of mercury poisoning, investigations have been carried out in one factory manufacturing physical apparatus, and in another where pharmaceutical compounds of mercury were being prepared. The principal physical apparatus involving the use of mercury is the thermometer. Clinical thermometers are made from strips of glass tubing which are cut to the required length. Each of these lengths is opened out to form the blister by means of a gas flame and air pump; the bulb is then blown. The next process is that of filling the bulb by inverting the tubes in a bowl of mercury, and the tubes are then boiled to remove any pocket of air. The thermometers are then tested by standard comparisons. The tops of the stems are next sealed, the blister is contracted, the heads are rounded off, and the thermometer engraved. During the process the workmen are exposed to the vapour of mercury, hot and bright flickering flames, hydrofluoric acid fumes, coal gas from leaking pipes and glass dust. Mercury can always be found in cracks and crevices on work benches and floors.

Clinical Observations

Some 72 men were examined in the workshop where thermometers were being made, and 11 men in the chemical works making mercurial compounds. Slight degrees of erethism and tremor must always be a matter of opinion, and in the tables which follow the opinion of one of us (K. P.) is recorded. An attempt was made to record the tremor by asking each man to write his name and address on a piece of paper, and to draw two straight lines, both in a horizontal and vertical direction. Fig. 1 shows an example of the results which were obtained. Gingivitis and salivation are common in the working population and there was no proof that any given case was attributable to mercury. The mass observation in these works suggests that most of the workers were suffering from mild degrees of mercury poisoning, but in no individual were the symptoms so severe that they could be attributed

without doubt to mercury, nor were they such as to produce any disability in the men, or any inability to work: but it is undesirable that any section of the population should be mildly intoxicated by any substance, and as a result of this work it is hoped that it may be possible to prevent this.

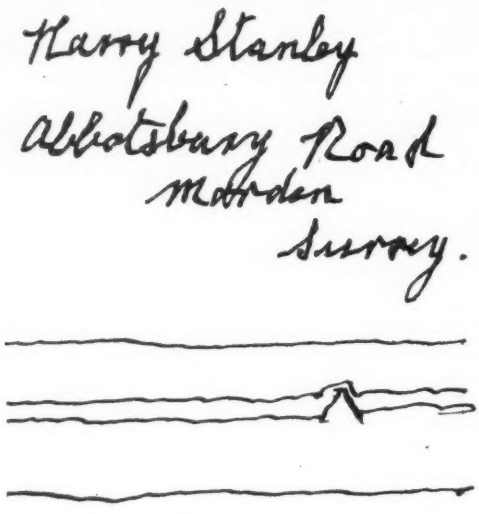

FIG. 1.—Specimen of handwriting and free-hand drawing of thermometer maker with mercurial tremor.

Groups of workers from each section of the factory were examined, and blood counts were taken. These revealed no abnormality, so that there is no confirmation of the reports by Dunajewsky and Peissakhovitch (1930) and Matussevic and Frumina (1936) that a lymphocytosis is a distinctive blood finding. Specimens of urine were collected over a 24-hours period at the week-end while the workers were clear of contamination in the factory (Tables 1, 2, 3, 4 and 5) and compared with specimens from 15 male controls taken from another factory where there was no contact with mercury (Table 6). These were examined quantitatively for mercury by the method of Milton (1946) the outline of which is as follows :

Principle. Organic matter was removed by oxidation and the mercury precipitated as sulphide using arsenic as co-precipitant. The mixed sulphides were separated from the solution and dissolved and the colour given by the mercury with a chloroform solution of dithizone was compared with that given by a standard mercury solution.

Reagents. Distilled water from all glass still.
Potassium permanganate.
Oxalic acid.
1 per cent. aqueous solution of sodium arsenite.
Hydrogen sulphide.
Concentrated nitric acid.
Concentrated hydrochloric acid.
Hydroxylamine hydrochloride.
10 per cent. ammonia.
Chloroform.
Dithizone (diphenylthiocarbazone).
Anhydrous sodium sulphate.
Sodium hypobromite solution.
Concentrated sulphuric acid.

Preparation of Dithizone Solution. It was found necessary to redistill all chloroform and ammonia before use; even the pure reagents contained traces of heavy metals.

Ten milligrammes of dithizone was dissolved in 50 ml. of redistilled chloroform and the solution shaken with 50 ml. of 10 per cent. ammonia. The chloroform layer was allowed to settle and drawn off. This separated most of the dithizone into the ammoniacal layer, leaving the impurities in the chloroform layer. Hydrochloric acid was added drop by drop to the aqueous solution until it was acid and no further precipitation of dithizone took place. The precipitate was dissolved in 100 ml. of chloroform and the chloroform drawn off; 20 ml. of this stock solution was diluted to 100 ml. with chloroform before use.

Calibration Graph. Using the Spekker Photoelectric Absorbtiometer a calibration graph was constructed from 0 to 50 μg. mercury. This calibration was repeated every time a fresh lot of any reagent came into use.

Blank. Fifteen ml. of the diluted dithizone solution was shaken with about 20 ml. of 10 per cent. ammonia and separated, and the process repeated with another 20 ml. of ammonia. The chloroform solution was dried by the addition of a little anhydrous sodium sulphate, and the colour read on the Spekker using the blue Mo.6 screen. In cases where the blank reading was more than 10 a fresh dithizone solution was prepared and the blank repeated.

Mercury Standards. A standard solution of mercuric nitrate, containing 50 mg. per cent. of mercury was made up. This was diluted 1 in 100 and from this standards containing, in 100 ml. of solution, from 5 to 50 μg. of mercury in 5 μg. steps were prepared. To each standard, in a separating funnel, 1 ml. of concentrated hydrochloric acid was added, then 3 ml. of dithizone solution and the funnel well shaken and allowed to stand. The chloroform layer was withdrawn into a 50-ml. graduated, stoppered cylinder. The extraction was repeated with four further 3-ml. portions of dithizone, making 15 ml. in all. The 15 ml. of dithizone extract was shaken with two 20-ml. portions of 10 per cent. ammonia to remove excess reagent, and dried with anhydrous sodium sulphate as in the case of the blank. The orange mercury-dithizone colour produced was read in the Spekker, and a graph constructed of Spekker reading minus blank against mercury concentration.

The method was developed for urine but could be adapted for use with blood or other tissues.

A 24-hour specimen of urine was collected and the volume recorded. Five hundred ml. of urine was refluxed with 20 ml. of concentrated sulphuric acid and 10 grams of potassium permanganate for three hours. While still warm 10 grams of oxalic

TABLE 1

CALIBRATING AND POINTING

Case No	Age	Duration of employment. Years	Erethism	Tremor	Stomatitis	Urine			Blood count						
						Volume in 24 hrs. ml.	μg 24 hrs.	p.p.m.	Hb.	R	W	P	L	M	E
1	17	2	—	—	—	—	—	—	116	6·0	7·8	65	28	5	1
2	17	3	—	—	—	—	—	—	119	6·3	11·8	70	25	5	—
3	28	15	+	+	—	2200	175	0·080	107	5·0	6·8	66	32	2	—
4	18	4	+	+	—	—	—	—	119	6·0	15·6	78	21	1	—
5	29	15	—	+	—	1040	278	0·267	114	5·6	12·8	62	33	4	1
6	29	15	—	—	+	1600	136	0·085	118	6·0	16·8	56	37	7	—
7	25	11	—	+	—	—	—	—	114	5·4	6·0	64	24	3	9

TABLE 2

CONTRACTORS

Case No.	Age	Duration of employment. Years	Erethism	Tremor	Stomatitis	Urine			Blood count						
						Volume in 24 hrs. ml.	μg 24 hrs.	p.p.m.	Hb.	R	W	P	L	M	E
8	17	3	+	+	—	2350	2260	0·961	121	6·0	7·6	58	36	4	2
9	39	25	—	++	+	1300	500	0·385	116	5·4	15·2	68	30	1	1
10	16	2	—	+	—	2300	3478	1·509	108	5·4	13·0	66	32	2	—
11	17	2	—	+	—	1500	866	0·577	112	5·7	12·2	64	32	3	1
12	18	3	—	+	+	1500	650	0·433	111	5·6	10·0	65	30	5	—
13	23	6	+	++	+	—	—	—	114	5·6	8·4	60	30	5	—
14	30	16	+	++	—	—	—	—	107	5·4	9·8	68	30	2	—
15	29	15	—	+	+	—	—	—	110	5·3	7·0	58	30	10	2
16	39	5	—	+	+	—	—	—	106	4·9	6·2	50	41	5	4
17	31	12	—	+	—	—	—	—	117	5·4	16·0	50	45	4	1
18	30	16	—	++	+	—	—	—	115	5·1	12·0	67	24	9	—
19	30	16	—	—	—	1600	900	0·562	106	5·2	17·2	57	34	3	—
20	28	14	—	+	+	2000	900	0·450	115	5·6	11·9	57	39	3	1
21	39	19	++	+	—	—	—	—	108	5·5	8·6	62	34	2	1
22	31	12	—	++	+	—	—	—	107	5·2	9·0	63	31	4	1
23	31	17	—	+	+	1800	540	0·300	100	5·0	9·2	58	36	5	1
24	33	6	—	+	+	1700	340	0·200	114	5·7	15·8	64	52	4	1
25	30	16	—	+	—	2500	300	0·120	116	5·8	16·0	70	26	3	1
26	40	26	+	++	+	2300	380	0·165	114	5·7	8·0	63	33	4	3

TABLE 3

HEAT TREATMENT AND BOILING IN

Case No.	Age	Duration of employment. Years	Erethism	Tremor	Stomatitis	Urine			Blood Counts						
						Volume in 24 hrs. ml.	μg 24 hrs.	p.p.m.	Hb.	R	W	P	L	M	E
27	41	12	+++	++—	—	2240	2280	1·017	108	5·4	15·6	59	36	5	1
28	56	1	+	+	—	1400	1200	0·857	97	5·8	11·8	56	37	5	1
29	18	4	+	+	—	880	2050	2·329	110	5·5	10·0	60	32	5	2
30	22	8	—	+	—	1550	1420	0·916	124	6·0	11·2	66	27	6	1
31	27	15	+	+	+	1230	1250	1·015	111	5·6	10·8	60	34	5	1
32	48	14	+	++	—	1500	1160	0·773	114	5·6	10·0	67	27	4	2

Haemoglobin by photo-electric alkaline haematin method 100 per cent. equivalent 13·8 grams per 100 ml.

TABLE 4

BLOWING AND FINISHING

Case No.	Age	Duration of employment. years	Erethism	Tremor	Stomatitis	Urine			Blood count						
						Volume in 24 hrs. ml.	μg 24 hrs.	p.p.m.	Hb.	R	W	P	L	M	E
33	31	15	−	+	−	1300	730	0·561	116	6·0	8·1	70	26	4	—
34	25	10	−	+	−	1160	1700	1·465	116	5·6	8·0	67	30	3	—
35	27	13	−	+	−	900	800	0·976	119	5·8	16·1	66	32	2	—
36	44	12	+	+	−	1200	1440	1·200	107	5·4	11·2	62	29	6	2
37	38	17	−	+	+	2250	1350	0·600	101	5·2	13·2	60	34	3	3
38	32	18	−	−	+	2400	1200	0·500	124	5·8	5·6	56	39	4	1
39	25	11	−	+	−	1520	885	0·582	125	6·0	10·6	64	32	2	2
40	22	9	−	+	−	710	280	0·394	127	6·0	9·6	60	34	4	1
41	24	10	+	++	−	1240	2261	1·823	120	5·9	6·4	64	32	4	—
42	17	3	−	−	−	—	—	—	116	5·4	13·4	59	34	7	—
43	22	7	−	−	−	2050	1435	0·700	111	5·4	7·2	67	29	3	1
44	31	17	−	+	+	1300	695	0·535	125	6·2	13·9	54	43	2	1
45	39	17	+	+	+	2290	1250	0·546	111	4·9	5·4	58	36	5	1
46	44	30	−	+	+	1200	540	0·450	116	5·9	6·2	69	25	4	1
47	29	15	−	+	−	1760	235	0·133	100	4·8	8·0	66	31	3	—
48	28	13	+	+	+	1100	643	0·585	118	6·4	6·4	67	29	3	1
49	28	14	−	+	−	1940	545	0·282	123	6·0	12·6	64	33	1	1
50	32	15	++	+	−	2460	2360	0·959	119	5·6	11·8	59	35	5	1
51	16	2	+	+	−	—	—	—	117	6·0	12·6	69	25	6	1
52	22	5	−	−	−	—	—	—	120	6·0	4·5	58	38	3	1
53	16	1	−	−	−	—	—	—	110	5·5	8·2	64	32	3	1
54	25	10	−	+	−	1220	528	0·432	119	6·1	12·0	66	30	4	¾
55	25	10	−	+	−	910	213	0·234	116	5·8	5·6	54	39	4	2
56	18	3	+	−	−	1110	1775	1·600	112	5·4	12·3	66	30	3	1
57	18	2	−	+	−	2600	1680	0·646	114	5·6	6·6	59	36	4	1
58	15	1	−	−	−	1190	1145	0·962	108	5·5	8·6	57	36	5	2
59	15	1	+	−	−	1700	276	0·162	111	5·6	10·2	54	40	4	2
60	16	2	+	−	−	—	—	—	103	5·2	11·3	61	33	5	—
61	16	1	−	−	−	1715	885	0·518	108	5·6	8·9	—	—	—	—
62	16	2	−	−	−	—	—	—	110	5·0	7·2	60	34	6	—
63	18	2	+	−	−	—	—	—	103	5·4	7·6	65	30	4	1
64	42	28	−	−	−	1850	142	0·077	119	5·5	7·8	71	22	4	3
65	49	6	−	−	−	1190	2110	1·771	105	5·6	6·8	64	29	4	2

TABLE 5

GLASS BLOWING AND BENDING

Case No.	Age	Duration of employment. Years	Erethism	Tremor	Stomatitis	Urine			Blood count						
						Volume in 24 hrs. ml.	μg 24 hrs.	p.p.m.	Hb.	R	W	P	L	M	E
66	23	8	−	+	+	—	—	—	112	5·5	12·0	62	31	7	—
67	22	7	−	−	−	—	—	—	111	5·4	9·3	58	37	5	—
68	30	14	−	+	+	—	—	—	99	5·1	11·6	46	46	7	1
69	27	3	−	+	−	—	—	—	116	5·9	8·2	60	35	4	1
70	22	8	−	−	−	—	—	—	90	4·5	7·9	59	36	5	—
71	16	2	−	−	−	1450	1090	0·751	120	6·4	6·4	62	33	3	—
72	18	3	−	+	+	1310	264	0·201	127	5·9	5·6	65	25	5	3

acid was added slowly to clear the solution. To the cold solution 1 ml. of sodium arsenite solution was added, and a slow stream of hydrogen sulphide passed through for twenty minutes. The mixed sulphide precipitate was coagulated by bringing the mixture to the boil. The mixture was allowed to stand overnight, and the clear supernatant fluid decanted. The precipitate was transferred to a centrifuge tube, spun out and washed twice with distilled water. Two ml. of concentrated nitric acid and 1 ml. of concentrated hydrochloric acid were added, and the tube warmed in a water bath to dissolve the precipitate. The solution was transferred to a separating funnel, and the volume made up to about 100 ml.; 5 gm. of hydroxylamine hydrochloride was added to remove the nitric acid which otherwise

interfered with the dithizone blank. One ml. of concentrated hydrochloric acid was then added and the extraction of mercury and colour comparison carried out as in the case of the standard. A blank run was carried out each time a fresh lot of permanganate came into use. This blank was subtracted from each determination. The results were expressed as micrograms per 24 hours' excretion.

It was also desirable to study the daily variability in the excretion of mercury, and one volunteer from the blowing and finishing shop (Patient No. 35) undertook to collect his urine daily for a whole week. The figures obtained were as follows:

	Volume in 24 hrs. ml.	μg 24 hrs.	*p.p.m.*
Thursday ..	2400	210	0·087
Friday ..	2400	290	0·121
Saturday ..	2700	96	0·035
Sunday ..	2000	96	0·048
Monday ..	1900	1000	0·526
Tuesday ..	1700	160	0·094
and on a second occasion:			
Monday ..	1100	148	0·134
Tuesday ..	1400	140	0·100
Wednesday ..	1600	152	0·095
Thursday ..	1500	157	0·104
Friday ..	1100	66	0·060
Saturday ..	1750	270	0·154

It was considered that this shows there is a considerable variability of the amount of mercury excreted in 24 hours, but that it is of the order which might be expected.

TABLE 6

CONTROLS

	Volume in 24 hrs. ml.	μg 24 hrs.	p.p.m.
A	2250	23	0·010
B	750	12	0·016
C	2500	50	0·020
D	2600	65	0·025
E	1800	9	0·005
F	1950	48	0·025
G	2000	40	0·020
H	1000	5	0·005
I	1000	10	0·010
J	1800	90	0·050
K	1000	20	0·020
L	2000	10	0·005
M	750	30	0·040
N	1000	29	0·029

An employee who had given long service to the firm became disgruntled, and decided to leave, for no clear reason. However, he was excreting a large amount of mercury in his urine, and it is highly possible that the act was a manifestation of erethism. Advantage was taken of the event to study the excretion of mercury after he had ceased to be exposed to it, and a 24-hour specimen was obtained 17 days after he left, when the excretion was found to be still present, though considerably lower.

Patient No. 50	Volume	μg/24 hrs.	p.p.m.
At work ..	2460	2360	0·959
17 days after leaving ..	2132	1290	0·605

Environmental Observations

The thermometer works was a long rectangular shop, 190 feet by 29 feet 6 inches, and of double width for the last 96 feet. It had a cubic capacity of 126,225 cubic feet. It was equipped with ventilators in the roof, and electric fans, and the number of air changes per hour was found to be slightly over two. At the near end the glass was cut into strips and blown, but no mercury was handled. In the middle the blowing and finishing were carried out, and the mercury added. At the far end the processes of boiling in and contracting, as well as calibrating, were performed. In a side room, three-quarters of the way down the workshop, the mercury was stored, and heat treatment of the thermometer was carried out.

A number of estimations of the amount of mercury in the atmosphere of the shop were made, using the method of Milton (1946).

Atmospheric Estimation

Mercury Vapour. A sodium hypobromite solution was prepared by dissolving 5 ml. of bromine in 20 ml. of 10 per cent. sodium hydroxide solution; 15 ml. of this solution was placed in a bead-bubbler, and the atmosphere to be examined was sucked through the solution at 2 litres per minute until about 200 litres had been sampled. The exact volume of the sample was recorded. The contents of the bubbler were drained into a separating funnel and acidified with hydrochloric acid. The bromine liberated was removed by adding hydroxylamine hydrochloride, two to three grams, until the bromine colour disappeared; 1 ml. of concentrated hydrochloric acid was then added and the extraction of mercury and colour comparison carried out as in the case of the standards above.

Mercury Salts. To determine the amount of mercury present as particulate matter, a No. 42 Whatman filter paper in a cone-shaped bakelite holder was attached to the inlet tube of the bubbler. The paper was afterwards pulped in about 200 ml. of distilled water and oxidized as for urine (see below). The contents of the bubbler were treated as in the case of mercury vapour.

These estimations were carried out under different

TABLE 7

Sampling point	Date	Temp.	Concentration Hg
Glass-blowing benches (remote from Hg. working).	28.2.45	—	25 μg/m³
	29.8.45	—	25 ,,
Boiling in bench (continuous Hg working).	28.2.45	—	100 ,,
	10.3.45	17·5° C.	10 ,,
	22.3.45	20° C.	108 ,,
	29.8.45	—	42 ,,
Treating room (Hg centrifuged into thermometer stems)	17.1.45	—	17 ,,
	28.2.42	—	175 ,,
	10.3.45	19° C.	70 ,,
	22.3.45	23° C.	670 ,,
	29.8.45	—	220 ,,

conditions of temperature, humidity, and wind direction, and show considerable variations. The concentration is increased in warm weather, and this is not surprising since air saturated with mercury vapour contains 1·84 p.p.m. at 20° C. and 8·5 p.p.m. at 40° C. These figures therefore indicate that the concentration of mercury is well below the saturation level, and that considerable variation in concentration could occur with local change in atmospheric conditions.

From these figures, however, it is clear that the workers are excreting far more mercury in their urine than they could possibly breathe in during a day. Assuming that a man breathes 10 cubic metres in a day, and he works in the mercury atmosphere for eight hours, the maximum mercury he could breathe in would be 356 μg. In the centrifuging room the atmosphere figures were higher, but even here it is extremely doubtful if the mercury excreted could be obtained from the air. In order to investigate the possibility of mercury being absorbed in other ways, bundles of finished thermometers were obtained and thoroughly washed in 20 per cent. ammonia. The washings were then estimated for mercury. Bundles of 50 thermometers taken at random were found to have on their surfaces the following quantities of mercury:

675 μg, 550 μg, 100 μg, 270 μg, 550 μg, 230 μg, 150 μg, 40,000 μg, and 580 μg.

The workers on the heat treatment and ' boiling in ' processes were asked to wash their hands at the end of the day, and the washings were examined for mercury with the following results:

TABLE 8

MERCURY IN μg FROM WORKERS' HAND WASHINGS

Patient No.	First visit	Second visit
27	9800 μg	3200 μg
28	3600 ,,	1680 ,,
29	7400 ,,	6400 ,,
30	—	7000 ,,
31	3200 ,,	7500 ,,
32	200 ,,	2500 ,,
34	920 ,,	—
37	800 ,,	—
41	600 ,,	—
73	2700 ,,	—

It is well known that mercury can be absorbed either through the respiratory tract, the alimentary tract or through the skin. All the evidence collected and put forward here, however, seems to establish that in thermometer workshops the absorption is principally through the skin, or alimentary track, and to a lesser degree through the respiratory tract.

Investigation at Chemical Factory

By contrast, the chemical factory was engaged in the manufacture of mercury salts. One section was devoted to the manufacture of mercuric and mercurous chlorides by the direct combination of mercury and chlorides under the influence of heat. The resultant salt was crushed and packed, and a fine particulate dust of mercuric chloride pervaded the atmosphere.

A second section of the factory was devoted to the manufacture of mercury oxides and iodides. This process consisted of treating the chlorides with alkalis or potassium iodide in the wet condition. The resultant precipitate was filtered and dried in ovens. Subsequent crushing and sieving of the dried salts was a dusty process and quite high concentrations of mercury were found in the atmosphere. Beside the main room there was a small weighing booth in which stood large, open dishes of mercury metal. The workers in this factory used sweat rags to keep the dust from the respiratory tract, but examination of the nostrils showed that much dust penetrated this protection.

TABLE 9

Sampling point	Date	Concentration of Hg
Weighing booth (Hg vapour).	21.11.44 28.11.44	185 μg/m³ 290 ,,
Process room (Hg oxides and iodides manufactured).	21.11.44 28.11.44	400 ,, 280 ,,
Sieving room (Hg oxides dried, washed and sieved)	21.11.44 28.11.44	1300 ,, 18,000 ,,
Sublimate room (manufacture).	20.12.44	380 ,,
Sublimate room (crushing and packing).	20.12.44	670 ,,

However, the amount excreted in the urine of the workers was, on the whole, less. The symptoms were also less well marked. Whether this indicates a different reaction to mercury metal as compared with the salts of mercury, or whether it is simply related to the amount absorbed, it is not possible to say.

It must be remembered also that calomel is made as a pharmaceutical preparation to be taken by mouth, and that a large number of the population take a considerable amount in this way, and yet never suffer from chronic mercurialism.

Prevention

The evidence of Neal and his colleagues (1941) that mercury poisoning does not occur if the concentration of mercury in the atmosphere is less than 100 μg per cu. m., together with the fact that men in the thermometer shop were excreting more mercury than could be absorbed from the air, indicates that, in these shops, measures should be directed towards cleanliness. Adequate washing facilities must be provided, and there must be strict supervision to ensure that the workers cleanse their hands and skin fully after each shift and before all meals. Mercury can seep into crevices, penetrate wooden floors, collect in interstices, mix with dust or amalgamate with solid metals. It vaporizes at room temperature and, therefore, potentially

TABLE 10

WORKERS IN CHEMICAL FACTORY

Case No.	Age	Duration of employment. Years	Erethism	Tremor	Stomatitis	Urine			Blood count						
						Volume in 24 hrs. ml.	μg 24 hrs.	p.p.m.	Hb.	R	W	P	L	M	E
73	53	25	+	+	—	1000	520	0·520	100	5·7	6·2	67	31	—	1
74	52	33	—	—	—	1900	750	0·395	104	5·2	9·9	67	33	—	—
75	62	31	—	—	—	1900	520	0·273	112	5·5	10·5	66	30	1	—
76	36	5	—	—	—	2400	1360	0·566	106	5·3	7·9	73	24	—	3
77	46	21	—	+	—	1400	530	0·378	108	5·4	10·0	63	33	—	3
78	54	5	—	—	+	—	—	—	100	5·1	6·3	72	23	1	1
79	38	5	—	—	—	1700	450	0·264	114	5·7	8·1	76	21	—	3
80	25	5	—	+	+	2400	590	0·249	114	5·7	8·5	66	33	—	1
81	54	½	—	—	—	—	—	—	—	—	—	—	—	—	—
82	46	3	—	—	+	2200	640	0·291	100	5·3	7·7	61	37	—	1
83	51	40	—	+	+	—	—	—	100	5·0	5·5	56	42	—	2

hazardous concentrations may occur in the atmosphere from seemingly negligible spillage. Wooden construction should be avoided; floors of concrete or composition material should be maintained without cracks or open interstices. Benches should be tilted so that mercury can be drained and collected, thus preventing vaporization and contact with the skin; wherever possible mercury should be used in closed procedures. Ventilation is a difficult problem and the mistake of forcibly ventilating open rooms must be avoided, since it increases the vaporization of mercury. Exhausts should be placed to draw off the air from apparatus where mercury is used, and the work space should be supplied with fresh air from an outside source. Concentrations of mercury in the atmosphere should never rise above 75 μg per cu. m. whatever the weather conditions, and mercury vapour detector apparatus is now available (Woodson 1939).

Summary

The symptoms of chronic mercury poisoning are erethism, tremor and stomatitis. Seventy-two men in a thermometer workshop and 11 in a chemical works making pharmaceutical compounds of mercury were examined. Mass observation suggests that the thermometer workers suffered from mild chronic mercury poisoning, whereas little abnormality was seen in the older group of chemical workers. Atmosphere and urine estimations showed that the thermometer makers were excreting up to 10 times as much mercury in a day as could possibly be absorbed from the atmosphere. Washings from bundles of finished thermometers and from the workers' hands at the end of the day suggest that this is to be explained by absorption of metallic mercury through the skin, or ingestion through the alimentary tract.

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BRONCHIOLITIS RESULTING FROM THE HANDLING OF BAGASSE

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Bagasse is sugar cane after the sugar has been extracted. Because of the toughness of its fibres and their good insulating properties it is used for making boards for interior decorating and for thermal insulating purposes. The sugar cane which is used for this purpose is grown in Louisiana and shipped to to this country in bales. When the bales are packed in America the bagasse contains 4 per cent. of glucose, but on arrival in England it is sugar free.

Until the outbreak of war the bales were broken by a wet process: they were placed under water in a soak pit, broken, and then carried by a conveyor belt from the small house in which this process was carried out to the main factory for processing. At the end of 1939, in order to save shipping space, the bales were packed more firmly and it proved no longer possible to break them in this wet process. A machine, known as a shredder, was therefore introduced to break the bales. This consisted of a wheel with multiple blades which revolved inside a case. The bales were first broken with a pick-axe and the sections then thrown into the case. The bagasse was thus shredded by this dry process and the small pieces were carried by the conveyor belt as before. This process proved to be very dusty. Bagasse, as found at the factory, consists mostly of cellulose fibre, though some of the cellulose is present in crystalline form. It also contains 1 per cent. of protein. Nagelschmidt found that bagasse gave 3 per cent. of ash on ashing at 400° C. By alternating treatment of this ash with cold dilute acid and hot dilute alkalies, over 80 per cent. could be dissolved. This dissolved portion contained the amorphous silica which came to about 50 per cent. of the total ash. The residue contained much quartz. Microscopic and x-ray diffraction analysis put the quartz at about 3–5 per cent. of the total ash. This amorphous silica formed roughly 1–2 per cent. of the bagasse and quartz 0·1–0·2 per cent.; many of the quartz particles were 20–30μ in size. It was most improbable, therefore, that any risk of silicosis could arise in handling bagasse. Bagasse was also found to contain many fungi. J. T. Duncan examined samples and also dust from the shredder room. He found many fungi present, and estimated that one gramme of the airborne dust contained 240 million fungal spores. These consisted of the uncultivable teliospores of fungi of the order Pucciniales (Rusts), as well as the conidia of many species of saprophytic fungi. About 20 different species were isolated in culture, including *Paecilomyces varioti*, *Aspergillus fumigatus*, *A. niger*, *A. terreus*, and *A. candida*, *Trichoderma lignorum*, *Monilia sitophilia*, *Aleurisma sp.* and species of Penicillium, Mucor, Rhizopus, etc.

In 1940, after the installation of the dry bale breaking machinery, cases of respiratory illness began to appear among employees working the shredding machine and breaking the bales. These cases were observed by Lloyd (1940) at the Brompton Hospital. He considered that he was dealing with an unusual kind of pneumonia which was related to bagasse, but did not think that there was sufficient evidence to establish the aetiology. He consulted E. L. Middleton, H.M. Medical Inspector of Factories who advised that the machinery should be modified by a jet of water being directed on to the wheel to allay the dust. This advice was carried out in 1941, and until 1944 no further case of respiratory disease which could be attributed to the bagasse occurred in England.

Review of Literature

Jamison and Hopkins (1941) described the case of a young negro labourer who was employed in 1940 unloading bagasse from a ship at a board-making factory in New Orleans. This was a very dusty occupation. He developed an acute febrile illness with marked dyspnoea, cough and scanty blood-stained sputum. Physical signs showed râles scattered throughout both lungs. X-rays showed 'miliary mottling' through both lung fields, but these cleared in the course of 2 months, leaving a normal healthy picture. The patient recovered completely. From a concentrate of a 24-hour specimen of his sputum a fungus was grown on two occasions. The authors, however, while stating that in their opinion this was the cause of the illness, fail to state the nature of the fungus.

Castleden and Hamilton-Paterson (1942) reported four case of 'bagassosis,' an industrial lung disease. The first was a boy of 19, who after working for six weeks in the spring of 1941, developed a bilateral pneumonia which radiologically showed an extensive consolidation in both lungs with enlarged mediastinal glands. The appearances did not show the typical miliary mottling of the syndrome associated with bagasse, but the process may have become confluent in this particular case, though it is impossible to distinguish from the atypical pneumonia which was prevalent in that year, and which has been well described by Drew, Samuel and Ball (1943), among others. Case 2 was an electrical mechanic aged 37, who was not himself engaged on

the work of breaking the bales. The machinery, however, was new and there were frequent breakdowns. He therefore spent considerable time in the house effecting repairs. X-ray of his chest in 1940 showed no abnormality beyond a catarrhal bronchitis. However, since that time he has developed an extensive progressive fibrosis of both lungs, He subsequently died in 1944 and an account of the necropsy is given later in this paper. (Case 12.) Case 3 showed no radiological changes in the lungs, and case 4 was a man aged 44 who had previously worked for 17 years in the Durham coalfield and showed the x-ray appearances which have been termed reticulation by Hart and Aslett (1942). Castleden and Hamilton-Paterson also described skin tests which they had carried out on some of the workers. These showed that some developed a skin sensitivity to extracts of bagasse. Sodeman and Pullen (1944) repeated these tests in persons exposed to bagasse, and in a group who had not been exposed. They failed to find a negative reactor in either group. Castleden and Hamilton-Paterson therefore left the disease in a very obscure position, especially as the third and fourth cases show no disease that can be clearly attributed to bagasse and the fourth man had a pneumonokoniosis caused by his work as a collier.

A further case was reported from New Orleans in 1944 by Jamison, Bryan and Day in a negro man aged 32, who had been loading railway freight cars for a month. He developed dyspnoea and x-ray of his chest revealed infiltration with a miliary appearance which cleared completely in the course of four months. No fungi were found in this case.

Finally, Sodeman and Pullen (1943) gave an account of a fatal case and (1944) an account of 11 further cases. They say that shortness of breath was invariably the chief complaint, but that cough was an early and important symptom. In four instances haemoptysis occurred: it was variable in amount, but lasted only a few days. The sputum was scanty and mucoid. Intermittent fever with temperature rising to 102° F. was observed and persisted up to three or four weeks. X-ray examination of the chest showed a miliary mottling throughout both lungs, most dense in the hilar areas in all cases. White cell counts were over 10,000 in 10 instances with a polymorphonuclear leucocytosis. Needle biopsy specimens were obtained from the lung of one patient in the sixth week of the disease,

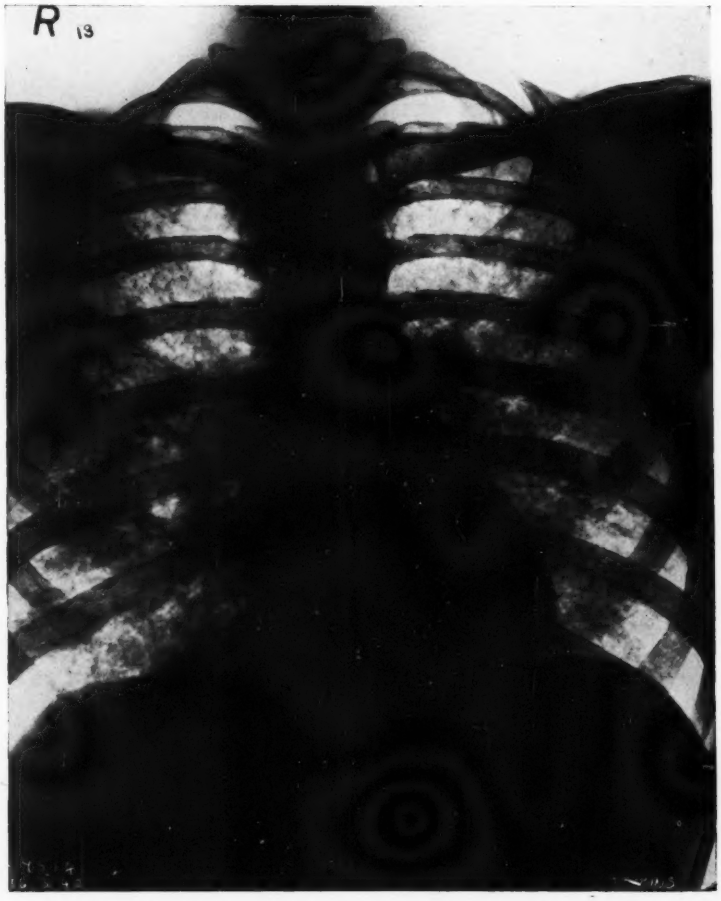

FIG. 1.—Case 1 in acute stage showing diffuse mottling with scattered confluent areas.

and sections were taken at necropsy from the lung of the patient who died. These showed pulmonary tissue with several 'spicules' of an irregular foreign material embedded in it. There was a fibroblastic reaction of the interstitial tissue of the lung. There were many large cells with a foamy cytoplasm in the alveolar spaces. The foreign bodies were microscopically similar to bagasse, and under the polarizing microscope these 'spicules' were seen to rotate the plane of polarized light; many smaller scattered pieces with an average diameter of 2·8μ were also seen.

Present Investigation

An attempt has been made to ascertain what has happened to all the men who worked on the bagasse shredder at the factory in England. It was discovered that in 1940 14 men were employed on this machine for continuous periods of more than 3 days; in 1941 20 men, and in 1942 10 men. These men worked in pairs for two weeks by day, followed by a two weeks' night shift. The work was considerably more dusty at night since blackout regulations necessitated the closing of the large doors to the shed which housed the machine. Of the 14 men employed in 1940, 13 were traced; of the 20 in 1941, 18 were traced; and of the 10 in 1942, 8 were traced. As the machine was in an experimental stage in 1940 the assistant works manager, the chemist, the personnel manager, an electrical mechanic and a carpenter also spent a considerable amount of time in the shed. All five of these have been traced and examined. Of 13 men traced who worked on the shredder in 1940 7 showed a characteristic acute respiratory illness, similar to that already described by Lloyd at the Brompton Hospital and the American workers; and of the 8 men employed during the first three months of 1941 3 showed signs of this illness.

Thus, out of 21 men employed on the shredder in a period of 15 months, 10 (47·5 per cent.) developed the illness. The onset of symptoms usually occurred after the men had been working on the machine for 8 weeks. The disease manifested itself as an acute febrile illness with extreme shortness of breath, cough with scanty black, stringy sputum and occasional haemoptysis. Signs were scattered throughout both lungs, and x-rays of the chest showed miliary shadows throughout both lung fields. The appearances

FIG. 2.—Case 1, three years after the acute illness.

FIG. 3.—Case 4 in acute stage showing extensive mottling with confluent areas.

were therefore those of an acute bronchiolitis. The patient's symptoms gradually improved over a period of six weeks, at the end of which time he had recovered and skiagrams showed the lung fields to be quite clear. One patient in 1941 died after 25 days' illness, but unfortunately no necropsy was performed. Except for the electrical mechanic, the five men of the technical staff showed no evidence of disease in their lungs at this time. The electrical mechanic (case 2 Castleden and Hamilton-Paterson, case 12 this paper) had repeated acute respiratory illnesses which progressed to a chronic condition. He finally died in 1944, and necropsy revealed chronic bronchiolitis and bronchiectasis. These cases give strong evidence that a specific disease manifesting itself by acute bronchiolitis, collapse, and pneumonia has occurred in the lungs of men who handle bagasse. The case histories of the ten men described above who showed specific disease follow.

Case 1. G. J., aged 28. He had worked for one year in the South Wales coalfield, three years in a rubber factory, and six years in the regular Army before joining the firm handling the bagasse in June 1938 as a labourer. He worked on the shredding machine from December 1st, 1939, to January 29th, 1940, when he was taken ill. He was admitted to the Brompton Hospital on March 16th, 1940, under Dr. W. E. Lloyd, complaining of shortness of breath, cough with purulent sputum, which was streaked with blood on one occasion, sweating and loss of weight. On examination he had a fever to 101° F., which persisted for three weeks. There were impaired percussion note and many coarse râles scattered throughout both lungs. X-ray of his chest showed a diffuse mottling with some confluent areas scattered throughout both lungs. There was considerable hilar enlargement (fig. 1). Further x-rays taken on April 15th, May 6th and July 20th showed that the process rapidly cleared. On July 15th, 1943, he was well, but still suffered from some shortness of breath and cough. He had no abnormal physical signs and x-ray of his chest revealed no abnormality (fig. 2).

Case 2. W. D., aged 65. He had worked as a newspaper roundsman, a dock labourer, in the Merchant Navy, in the Army, and as a builder's labourer before joining the firm in September 1937. He worked as a labourer until December 1939, but in that month and January 1940, when he was taken ill, he worked on the shredding machine. He was admitted to the Central Middlesex Hospital under Dr. H. Joules on April 25th, 1940, complaining of shortness of breath, cough with a

FIG. 4.—Case 4; three years after the acute illness.

considerable amount of sticky sputum, and progressive loss of weight. On examination his apex beat was not displaced, his pulse rate 88, blood pressure 150/105. There was slight thickening of the peripheral arteries and his aortic second sound was accentuated. He was cyanosed and dyspnoeic; the percussion note was normal and the breath sounds vesicular. Crepitant râles and occasional rhonchi were heard all over the chest. His sputum was negative for tubercle bacilli and lung fibres. The blood sedimentation rate was 40, and his blood count showed no abnormality. X-ray of his chest on April 27th, 1940, showed a diffuse miliary mottling scattered throughout both lung fields. By May 24th, 1940, this had substantially cleared, and on June 4th, 1940, he was discharged. Further x-rays taken on September 5th, 1940, and July 15th, 1943, revealed no abnormality, although he still complained of a severe winter cough with thick black sputum, and on examination there were scattered crepitations throughout both lungs.

Case 3. T. H., aged 29. He had worked as a pithead worker in Scotland for 5 years, and had then been unemployed until he joined the firm on January 21st, 1940. He worked as a labourer until March 16th, 1941, and then worked on the shredding machine until he was taken ill on April 27th, 1941. He was extremely short of breath, and had a cough with black stringy sputum; no haemoptysis and no pain. He was in bed for 3 weeks with a temperature up to 103° F. for 3 days. An x-ray was taken of his chest at Wembley Hospital on May 29th, 1941, and this showed mottled opacities scattered throughout both lungs, their frequency being greater on the right side. On September 23rd, 1943, he was quite well, working at the degreasing of metal sparking plugs. He had no symptoms, no abnormal physical signs, and an x-ray showed no evidence of disease in the lungs.

Case 4. A. W., aged 54. He had worked as a farm labourer and in the Army until he joined the firm on July 9th, 1940. He then worked on the shredding machine until August 22nd, 1940, when he was taken ill. He was admitted to St. Charles Hospital, Ladbroke Grove, on August 28th, 1940, complaining of severe cough with purulent sputum, shortness of breath and night sweats for two weeks. On examination there were scattered râles over both bases and rhonchi at the left apex. He had clubbing of the fingers. During his first week in hospital he had a fever up to 100·5° F. Examination of his sputum showed no tubercle bacilli. X-ray of his chest on August 28th (fig. 3) showed very extensive mottling with some confluent areas throughout both lungs. X-ray examination on September 30th, 1940, showed that this process had almost cleared, and he was

discharged from hospital on November 3rd, 1940. On July 15th, 1943, he was well, but still complained of some shortness of breath. On examination there were no abnormal physical signs. X-ray of his chest revealed no abnormality (fig. 4).

Case 5. S. B., aged 40. He had worked as a building labourer until he joined the firm on July 16th, 1940. He worked on the shredding machine from July 16th to September 1st, when he was taken ill. He was admitted to Willesden General Hospital under Dr. Pearse Williams, complaining of cough with a small amount of muco-purulent sputum, shortness of breath and sweating for 2 weeks, and slight recent loss of weight. There was no history of haemoptysis. On examination there was impaired percussion note and many râles throughout both upper zones. Sputum contained pus and showed numerous acid-fast bacilli. On culture streptococcus viridans, staphylococcus aureus and micrococcus catarrhalis were present. Dr. Rohan Williams reported on an x-ray taken on September 12th, 1940, as follows:—Bilateral diffuse parenchymal inflammatory changes in both mid and lower zones, the upper zones being relatively clear. Probably a diffuse bronchogenic tuberculosis. Dr. Rohan Williams suggested the possibility of an occupational cause for the appearances. The patient was seen by the Tuberculosis Officer who agreed that he was suffering from pulmonary tuberculosis, and he was admitted to Clare Hall Sanatorium in November 1940. His symptoms had disappeared by then. His sputum contained no tubercle bacilli, and an x-ray taken on November 20th, 1940, revealed no abnormality. He was examined on July 15th, 1943, when he was quite well, had no abnormal physical signs, and an x-ray of his chest revealed no abnormality.

Case 6. J. S., aged 45. He had worked on the roads and railways in Ireland, and for 10 years as a window cleaner in England before he started working on the shredding machine on October 12th, 1940. He continued to work on the machine until November 20th, 1940, when he was taken ill. He attended the Brompton Hospital on December 11th, 1940, complaining of severe shortness of breath, cough with much sticky purulent sputum, sweating, and loss of weight. On examination there were many scattered râles throughout both lungs. X-ray showed a diffuse soft mottling throughout both lungs with some confluent areas. He was admitted to the hospital from January 3rd, 1941 to February 19th, 1941, when he was found to have a low grade fever to 100° F., which quickly subsided. X-ray examination on February 19th showed that the changes had completely disappeared. On July 15th, 1943, he was well with no symptoms, no abnormal

FIG. 5.—Case 8 showing scattered mottling, confluent areas, and enlarged hilar glands.

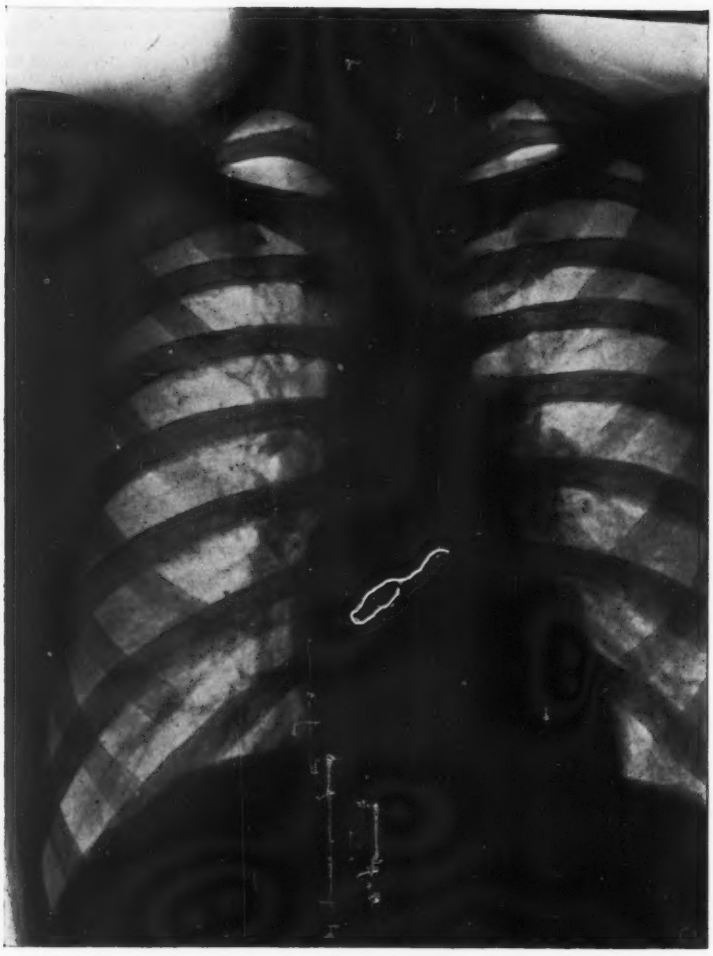

FIG. 6.—Case 8, three years after the acute illness.

physical signs, and there was nothing abnormal on x-ray examination.

Case 7. W. H., aged 38. He worked for 5 years as a collier's assistant in the South Wales coalfield, and for 15 years as a platelayer on the London, Midland and Scottish railway before joining the firm in October 1940. He worked from then until December 23rd, 1940, on the shredding machine when he was taken ill with fever, shortness of breath, cough with a small amount of sticky sputum, and loss of weight. He attended the Central Middlesex Hospital on February 27th, 1941, when he was found to have no abnormal physical signs. An x-ray was taken, and this revealed slight miliary mottling scattered throughout both lungs with enlargement of the hilar glands. On September 21st, 1943, he was in the Army. He had no symptoms, no abnormal physical signs, and x-ray examination of his chest showed no abnormality.

Case 8. E. P., aged 31. He worked in the motor engineering trade, in brickyards, and as a builder's labourer, before joining the firm as a labourer in October 1939. He worked on the shredding machine from September 1940 to January 25th, 1941, when he was taken ill. He attended the Northampton General Hospital under Dr. Eric Shaw on February 25th, 1941, complaining of cough with thick phlegm, shortness of breath, fever, and loss of weight. There were signs of consolidation at the base of the right lung. An x-ray showed scattered mottling throughout both lungs with some confluent areas and enlargement of hilar glands (fig. 5). He again attended on June 18th, 1941, when x-ray showed no abnormality. On July 4th, 1943, he still complained of much shortness of breath, cough and sputum, but an x-ray of his chest showed no abnormality in the lungs (fig. 6).

Case 9. W. W., aged 30. He had worked for a few months as an assistant collier in the South Wales coalfield, and then as a builder's labourer before joining the firm. He worked on the shredding machine from January 20th, 1941 to February 22nd, 1941, when he was taken ill. He was admitted to Wembley Hospital on March 6th, 1941, with severe shortness of breath and cyanosis. There were scattered patches of consolidation with bronchial breathing and coarse râles at both bases. On March 11th, 1941, there was a pleural friction rub over the right lower lobe. He was treated with oxygen at 6 litres a minute through a BLB mask, but the disease gradually spread and his condition deteriorated. He

was considered to be too ill for x-ray examination, and died on March 19th, 1941. There was no necropsy.

Case 10. S. N., aged 21. (Case 1, Castleden and Hamilton-Paterson, 1942.) He had worked as a fish-boy and milk roundsman before joining the firm as a labourer on September 11th, 1939. He worked on the shredding machine from March 21st, 1941, to April 21st, 1941, when he was taken ill. He was admitted to Redhill County Hospital under Dr. Castleden on May 4th with increasing shortness of breath and a cough with scanty sputum, sometimes blood-stained. His temperature was 100·6° F. on admission and he was febrile for 19 days. He was orthopnoeic and cyanosed: chest movement was poor, the percussion note was impaired, and there was distant bronchial breathing in small areas at both bases. The sputum contained M. catarrhalis, streptococcus viridans and fusiform bacilli. Staphylococcus aureus and pneumococcus Type 4 were each grown on one occasion. No tubercle bacilli were found. Blood count showed 4,610,000 red rells per c.mm., 78 per cent. haemoglobin, 11,300 leucocytes per c.mm., 83 per cent. neutrophils, 14 per cent. lymphocytes, 3 per cent. monocytes, and no eosinophils. X-ray on May 5th, 1941, showed extensive consolidation in both lower lobes with marked enlargement of the hilar glands. X-ray on June 15th showed some extension of the process, but on July 5th there was marked clearing, and by August 29th the lungs were completely clear. On August 27th, 1943, he was well and serving at sea with the Royal Navy. He had no symptoms, no abnormal physical signs, and x-ray of his chest showed no abnormality in the lungs.

In 1943 all the workers in the factory were interviewed and occupational histories taken. It was found that out of 163 men employed 22 had worked in the coal mines. A portable x-ray apparatus was taken to the factory and a skiagram (15 in. × 12 in.) was taken of the chest of all the workers. Of the 22 men who had previously been employed in the coal mines 19 showed evidence of chronic pulmonary disease, ranging through all stages of fibrosis, reticulation, nodulation and massive shadows. Three of these men, two showing fibrosis and one reticulation, had also worked on the shredding machine. These appearances could be superficially confused with those caused by bagasse, but whereas the latter is an acute febrile illness which clears in weeks or months, the former is a chronic state with no change over many years. Eight cases of pulmonary tuberculosis were also discovered.

Specimens of sputum obtained from the four men, who were working on the shredding machine at this time, were submitted to Duncan who isolated from them species of fungi common on the

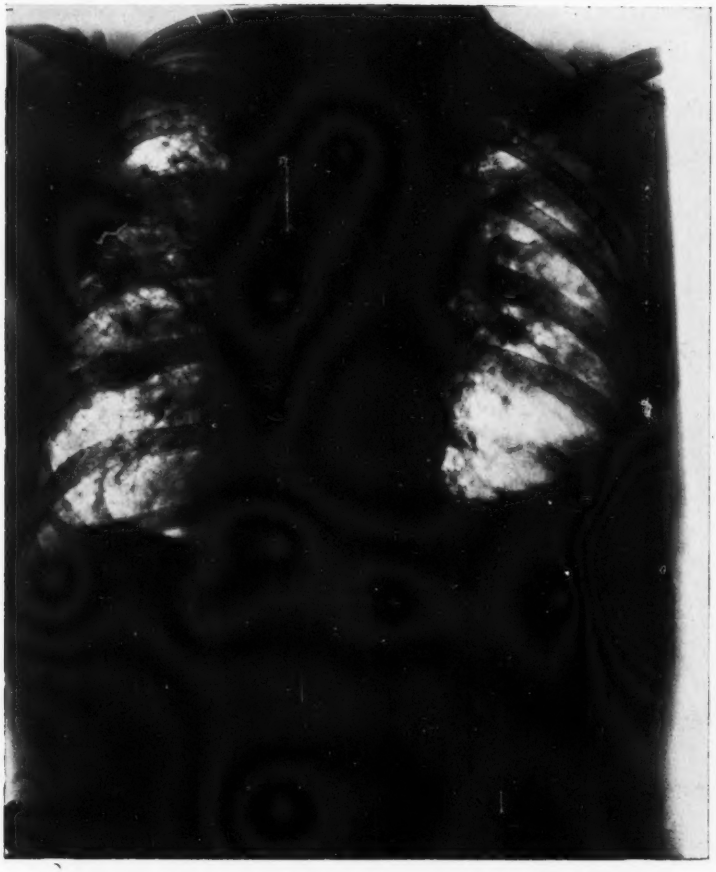

FIG. 7.—Case 12 showing extensive fibrosis 3 years after working with bagasse.

bagasse and in the dust of the shredder room. Microscopical examination of the sputum did not reveal evidence pointing to mycosis, but showed the ingredients of the airborne dust. The fungal elements present, from which the cultures were obtained, represented merely the result of recent inhalation of dust.

In 1944 the men working on the shredder again had skiagrams taken of their chests. One man complained of symptoms and the skiagram revealed the characteristic picture described in the previous cases. Attempts were made to persuade him to come into hospital but he declined, and being a citizen of Eire he returned to that country. As far as is known he was afebrile. It seems possible, therefore, that the x-ray changes may precede the onset of the acute illness and be caused by bronchiolar obstruction, giving rise to small areas of collapse.

Case 11. J. S., aged 50. Started work at the age of 15 as a messenger boy. Apart from 6 years in the Army he had worked in a bacon factory, and on buildings in Ireland until 1943 when he came to England and started work at the board-making factory. For the first 6 months he worked on the beater floor in the main building and for the 6 months previous to examination he worked on the shredding machine. For 3 months he had had shortness of breath on exertion, a severe dry cough which was worse at night and in the early morning. He had a poor appetite and was losing weight. On examination no abnormal physical signs were found. A skiagram of his chest showed a diffuse miliary mottling throughout the lung fields.

A man who had worked part of three shifts on the shredding machine but had otherwise worked for a year at the far end of the factory, was discovered on x-ray examination to have bilateral fibrosis and possibly cavitation of an unusual type in both lungs. The x-ray appearances were similar to those of the electrical mechanic already referred to as working in the shredder room in 1940. Both men were admitted to hospital for further investigation. Their case histories were as follows :—

Case 12. T. M., aged 38. (Case 2, Castleden and Hamilton-Paterson, 1942.) He had worked as a fitter until he joined the firm as an electrical maintenance mechanic. In this capacity he was intermittently in close contact with the dry crushing process during the installation of the new shredding machine between August 1939 and September 3rd, 1940, when he first complained of shortness of breath, cough and scanty sputum. His only previous illness was in 1935, when he was in Salisbury Infirmary for a few days with acute bronchitis.

FIG. 8.—Case 13 showing extensive pulmonary fibrosis.

A skiagram was not taken of his chest at this time. He attended the Willesden General Hospital in October 1940 and Dr. Rohan Williams reported on an x-ray of his chest. The appearances were consistent with a catarrhal bronchitis, but no further pulmonary lesion was noted. He returned to work in the office on October 30th, 1940, but was away sick with similar symptoms from November 22nd to December 10th, 1940. In December 1940 he began to be short of breath at work, and in January 1941 he was out of breath on the least exertion. On January 17th, 1941, he attended Redhill County Hospital under Dr. L. I. M. Castleden. He had clubbing of the fingers, and his chest was kyphotic with many fine râles in all areas of both lungs. X-ray showed a few apical scars and a little fibrosis in the right mid zone. In May 1941 he was admitted to Wembley Hospital where an x-ray was reported on as showing bilateral apical tuberculosis. The shortness of breath increased and he was admitted to Redhill County Hospital on November 22nd, 1941, and did not return to work at the factory handling bagasse. He was orthopnoeic but afebrile; the percussion note was impaired at the left apex with bronchial breathing and bronchophony. Crepitations were present throughout the left lung, and at the right apex. The sputum was mucoid, viscid, scanty, but never blood-stained. Polymorphs and lymphocytes were present. Haemolytic streptococci and pneumococci (type 23) were present. Tubercle bacilli were not found in the examination of 18 smears. A special search was made for fungi, but none were seen. A blood count showed 4,590,000 red cells per c.mm., 94 per cent. haemoglobin, 10,200 leucocytes per c.mm., 51 per cent. polymorphs, 32 per cent. lymphocytes, 6 per cent. monocytes, 11 per cent. eosinophils. X-rays showed extensive fibrosis in both upper lobes with multiple cavities. He was discharged on February 15th, 1942, with less dyspnoea. His condition remained unchanged and he was admitted to the London Hospital on August 16th, 1943, with orthopnoea and markedly clubbed fingers. There was poor movement of the left side of his chest with many scattered high-pitched rhonchi and râles. His sedimentation rate was 30 mm. in 1 hour (Wintrobe). His blood count showed 97 per cent. haemoglobin (alkaline haematin, photo-electric cell method) and 6,440 white cells per c.mm. X-ray of his chest (fig. 7) showed marked generalized fibrosis in both lungs and suggested the presence of cavities. His sputum showed no tubercle bacilli on smear, concentration, culture or guinea-pig inoculation. J. F. Duncan demonstrated the presence of *Candida sp.* On August 23rd Mr. Vernon Thompson performed a bronchoscopy which revealed no abnormality. He obtained bronchoscopic swabs from the pus present in the bronchi. No tubercle bacilli could be demonstrated in this pus either on smear, concentration, culture or guinea-pig inoculation. None of the fungi common in the sputum of men working on the shredding machine were found in the bronchoscopy specimens or any other specimens of sputum. The patient was treated with large doses of iodides without improvement, and discharged on November 27th.

He attended the out-patient department until September 23rd, 1944, when his breathlessness became very much worse, and he felt he had not the strength to cough up sputum. He was therefore admitted to hospital. On examination he was orthopnoeic, temperature 98°, pulse 110, respiration 32 at rest in bed. He was cyanosed and had a dry tongue with a brown fur and enlarged lymph glands in the upper cervical chain. There was a soft systolic murmur in the mitral area with a triple rhythm in the tricuspid area. His blood pressure was 105/70. Movement on both sides of the chest was poor and all accessory muscles of respiration were in play. There were palpable rhonchi over the front of the chest; percussion note was impaired at both apices; breath sounds were bronchial at both apices, but vesicular elsewhere. Coarse râles and rhonchi were heard all over the chest. His liver was felt 3 finger-breadths below the right costal margin. The sputum contained multiple organisms with streptococcus viridans and pneumococcus predominating. Blood count: 91 per cent. haemoglobin (photo-electric cell), 13,900 leucocytes, 90 per cent. polymorphs, 2 per cent. eosinophils, 7 per cent. lymphocytes, 1 per cent. large hyalines. X-ray of chest showed no marked difference from previous x-rays. His condition steadily deteriorated and he died on October 16th, 1944.

Necropsy revealed much thick pus and injection of mucosa in trachea and main bronchi. The posterior two-thirds of the upper lobe of the left lung was greatly contracted, airless, anthracotic and fibrotic, showing smooth rubbery, mottled grey and black cut surfaces and considerable bronchiectasis up to 0·5 cm. diameter with a few bronchiectatic cavities up to 1 cm. diameter. These cavities were empty and had smooth grey linings. There were thickened dilated bronchioles up to 0·3 cm. diameter throughout the anterior third of left upper lobe and throughout lower lobes of both lungs and right middle lobe, with much intervening well aerated emphysematous lung tissue. There were large areas of severe emphysema with emphysematous bullae in all borders of lower and middle lobes of both lungs. There were numerous irregularly shaped, grey and black mottled, tough mostly subpleural patches up to 3·5 cm. broad of completely fibrotic lung tissue with a smooth rubbery cut surface throughout the upper lobe of the right lung affecting all the lung tissue in the apex and almost all of the posterior fifth of the lobe, but with a considerable amount of well aerated intervening lung tissue in the rest of the lobe. Similar areas of fibrotic pneumonia formed an interrupted irregular subpleural zone 12 cm. long with depth varying from 0·2 to 1·2 cm. in the posterior part of each lower lobe, and also patches 1·5 cm. broad near the hilum of each lower lobe. Thick white pus was present in many bronchioles throughout both lungs. Numerous areas of fibrous thickening, up to 0·2 cm. thick, of the visceral pleura covered both lungs. Fibrous pleural adhesions obliterated both pleural cavities except over part of the left lower lobe.

Professor H. M. Turnbull is working on the pathology of the disease and when the work is completed will publish a full account of it in this journal.

Case 13. T. K., aged 42. He had worked on a farm and as a cellarman in Ireland before he joined the firm in July 1938. He worked in the packing department and on the 'defibrator' in the main factory, and on three occasions he worked part of a shift on the shredding machine. He complained of no symptoms, but in the mass radiographic examination of the employees on September 10th, 1943, he was found to have extensive fibrosis in the mid zone of both lungs (fig, 8). He was admitted to the London Hospital on November 23rd, 1943, and tomographs suggested the presence of cavities. Sputum examination revealed no tubercle bacilli on smear, concentration, culture or guinea-pig inoculation. Mr. Vernon Thompson performed a bronchoscopy on December 6th, 1943, and took swabs from the pus in the bronchi. Examination revealed no tubercle bacilli on smear, concentration, culture or guinea-pig inoculation. None of the fungi common in the sputum of men working on the shredding machine were found. The patient was discharged on December 8th, 1943, and his condition has not changed since then.

None of the fungi common in the sputum of men working in the shredding room were found in these two men's sputum. It may be presumed that when they and the other men were exposed to the dust of the shredding room, their sputum and probably the larger bronchi contained numerous inhaled fungus spores. We have no evidence of survival of any of the fungi in the lungs of men after removal from exposure to the atmosphere of the shredding room.

These two men were less exposed to bagasse than those who developed the acute infection described in cases 1–10; nevertheless they were exposed and

their histories and clinical investigations are not typical of any known disease of the lungs. Case 12 had an acute respiratory illness at the commencement and though no skiagrams are available of his lungs at that time the natural chronic sequel to an acute bronchiolitis and pneumonia would be chronic bronchiolitis and bronchiectasis, and this is the condition that was found in his lungs at necropsy. Pathologically this chronic bronchiolitis and bronchiectasis does not differ from that arising in any chronic interstitial pneumonia, but the distribution in this case involving the upper lobe particularly posteriorly, and causing tough subpleural patches of fibrotic lung was most unusual except in the diseases caused by dust. It seems probable, therefore, that it was a chronic sequel to disease in the lung caused by bagasse.

The two cases reported by Jamison and his colleagues (1941 and 1944), the twelve cases reported by Sodeman and Pullen (1943 and 1944), and the ten cases reported here are all similar, and show almost conclusively that bagasse dust can give rise to acute bronchiolitis and pneumonia. Out of the 24 cases there were two deaths, a mortality rate of 4·3 per cent. Here, therefore, is an industrial disease with serious complications.

The exact aetiology of the disease, however, remains obscure. There are undoubtedly a great many fungi present in bagasse and Duncan has recovered similar fungi from men who have worked on the shredding machine, but there is no evidence that any particular fungus has caused the syndrome which is described; neither is there any evidence that the disease has been caused by any specific bacterium or virus. It is to be noticed that acid-fast bacilli were found in the sputum in case 5, and the case described by Jamison and Hopkins (1941). It is possible that the disease is caused by the irritation of the dust itself in the bronchioles, giving rise to an inflammatory reaction, but this hardly seems a satisfactory explanation.

Fungi may play an important role in breaking down the fibre into a very fine vegetable dust, and may possibly even render this toxic. Fawcitt (1938) has described a condition which he labelled 'Farmer's lung.' This disease is caused by inhalation of a fine vegetable dust from mouldy hay and in its symptomatology and radiological findings it closely resembles byssinosis as well as the condition described in this paper as caused by bagasse. A similar disease occurs in horses and is known as 'broken wind.' These four diseases so closely resemble each other that they may possibly belong to a single group with a common pathology.

Neal, Schneiter and Caminita (1942) described an acute febrile illness occurring among workers exposed to high concentrations of stained cotton dust. Although these authors attributed this disease to the presence of a gram-negative rod shaped bacterium, it is possible that this disease also resulted from the fine vegetable dust created in the process, or from the breakdown of vegetable matter by the organism described.

Prevention

In the spring of 1941 the process of breaking the bagasse bales had been rendered wet by a spray of water to allay the dust. Exhaust ventilation was also installed. Since that period only one further case of the disease has been observed.

Summary

An account is given of 11 cases of acute bronchiolitis and pneumonia arising from the inhalation of bagasse dust. One of these cases was fatal. Severe dyspnoea, cough with scanty sputum and occasional haemoptysis were the usual symptoms. There was a characteristic x-ray appearance which showed a miliary mottling scattered throughout both lungs with heavy shadows at the hilum.

Reference is made to 14 similar cases described in the literature, among which there was also one death. The aetiology is uncertain but the disease was most likely caused by fine vegetable dust. Two workers who had been in contact with the bagasse developed extensive fibrosis of lung, and one of these subsequently died. Necropsy revealed chronic bronchiolitis and bronchiectasis.

The method of prevention suggested is to allay dust and to handle bagasse in a moist state. Exhaust ventilation is also desirable.

Acknowledgements

Our indebtedness to the many individuals and hospitals who have made this investigation possible is acknowledged throughout the text. We are also grateful to Drs. M. H. Jupe and D. Jennings for their help in interpreting the x-rays, and to Mr. H. Ferrier for carrying out the mass radiography.

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THE AETIOLOGY, PREVENTION AND TREATMENT OF CHRONIC BRONCHITIS*

BY

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No one can pretend that the treatment of chronic bronchitis at the present time is anything but profoundly unsatisfactory. No attempts are made at prevention, whilst treatment of the established case depends to a large extent upon the depth of the patient's purse, and consists of a bottle of cough mixture for the poor patient, and advice to winter in South Africa for his wealthier fellow-sufferer.

It is therefore quite clear that a reconsideration of the whole problem is urgently indicated, especially as this disease is well known to be an extremely common condition in England, and one of great economic importance, extending in most cases over many years of the patient's working life, and rendering him year by year progressively less capable of following his occupation. The final result is likely to be a state of miserable invalidism, the patient himself crippled by prohibitive shortness of breath, and both he and his family irritated beyond endurance by his constant ineffectual coughing.

Aetiology

Chronic bronchitis is essentially an inflammatory condition, and to bear this constantly in mind is of cardinal importance in the understanding of the problem under discussion. In all acute and chronic inflammatory disorders the nature, severity, and duration of the disease are determined by a balance between the virulence and numbers of the causative agent on the one hand, and the local and general resistance of the patient on the other. Chronic bronchitis is no exception. The organisms are known. They are bacterial, and include micrococcus catarrhalis, pneumococci, various forms of streptococci, both haemolytic and non-haemolytic, especially streptococcus viridans, together with Friedlander's bacillus, and staphylococci. The organisms are almost always mixed, and it is not possible to say that any of the above predominates in the majority of cases. The resistance of the patient depends upon a number of factors. It is probable that first and foremost amongst these is the natural inherited resistance of the individual. Some persons, indeed some whole families, are unduly prone to respiratory infection both of the upper and lower respiratory tract. Lowering of their general and local resistance to respiratory infection is brought about in these susceptible persons by a combination of causes; these include the obvious ones of chill, damp, fatigue, under-nutrition, and debility from a recent illness of some kind or other. No less important are the local factors: mouth breathing, excessive cigarette smoking, and any real focus of infection in the naso-pharynx. In addition, it is well known that workers in certain occupations are particularly likely to develop chronic bronchitis, and naturally these occupations are those involving the inhalation of dusts and fumes, especially those of silica, iron, steel, asbestos, carbon and kaolin; less commonly, the fumes of chlorine, ether, ammonia, nitric acid and, strange to relate, also in a few apparently innocuous trades, such as hairdressing.

There is an additional group of cases, in which an essential underlying feature is the presence of a permanent, infective focus in the lung, usually bronchiectatic, often with fibrosis, and not uncommonly to be found in stout, thick-chested subjects with a poor respiratory movement. In this minority upper respiratory infection is of less importance in producing chronicity, but is responsible for repeated symptomatic exacerbations.

Clinical Course

It is almost invariable to find that a patient with chronic bronchitis gives a history of recurrent acute or subacute attacks in the past. In many cases the bronchial mucosa is first damaged in childhood during one or other of the acute specific fevers, most often measles or whooping cough, especially if complicated by bronchopneumonia. At that time the bronchitis clears up quickly, but probably another attack will occur the next winter, after an upper respiratory infection, and on this occasion prove more stubborn, and when the same sort of thing happens the following winter, it lingers on for many weeks, and finally, in succeeding years, tends to drag on in a mild form throughout the whole winter. The ultimate result is a permanently damaged and denuded bronchial epithelium, which reacts violently to the slightest inhaled irritant, so that the sufferer is never really free from symptoms the whole year round. Nor is this all, for owing to the obstructive element, sooner or later in most cases will appear the twin bogies of chronic bronchitis: bronchitic asthma and emphysema.

Preventive Treatment

Any attempts at prevention must be made before the chronic inflammatory changes in the bronchial tree are finally established, i.e. at a fairly early stage, when the patient is still giving a history of recurrent autumnal or winter exacerbations, but is well during the rest of the year. Every effort must then be made to protect these susceptible individuals from upper respiratory infection and its effects. This might be accomplished in various ways, and from every point of view, not least the assessment of results, the measures to be suggested could best be applied to large groups of persons rather than individuals. These measures will consist of:

1. Attempts at diminishing the frequency of upper respiratory infection in those prone to develop bronchitis.
2. Attempts at protecting the individual from the effects upon his bronchial mucosa of an upper respiratory infection, should he contract one.

It is accepted that upper respiratory infections occur either by direct droplet spread from individual to individual, or by inhalation of airborne pathogens spread in the atmosphere from a more distant source. Although

* A paper read before the London group of the Association of Industrial Medical Officers in September 1945.

the former is probably the more common way of contracting an upper respiratory infection, the latter also accounts for a considerable number of cases, and its importance may well have been somewhat underestimated in the past.

Measures aimed at diminishing direct droplet infection have quite properly attracted a great deal of attention for a number of years past. The damage is done, of course, in crowded places, some at least of which can be avoided only with difficulty by most persons, for example the public transport vehicles. However, widespread propaganda against indiscriminate coughing and sneezing is obviously of great value, and it is evident that warning notices should be posted in all crowded places in public and industrial premises, and elsewhere. Bronchitic subjects might be well advised to walk or cycle to work if they can, or if travel is unavoidable, a bus is probably a better choice than the underground. In workrooms, proper spacing of workers is naturally essential and the erection of glass screens between opposite benches has much to recommend it. It is a wise course to send home anybody who arrives at work in the morning with a streaming head cold.

Measures aimed at controlling distant airborne droplet spread are important. Droplets are disseminated for a surprising distance by coughing, at least 15 feet, and they remain airborne for a considerable time. It has been found possible to recover influenza virus from various parts of a large room one hour after its suspension in the atmosphere. In order to prevent the spread of respiratory infection from these airborne droplets, the concentration of pathogens in the atmosphere must obviously be kept as low as possible. This might be achieved in various ways:

(i) Improved ventilation in indoor premises. Although most large buildings are equipped with some form of mechanical ventilation, natural ventilation can usually be made satisfactory by attention to various elementary points. Natural ventilation depends upon wind pressure and gravity, air being driven in through windows on the windward side and out on the leeward. Gravity effects are due to temperature differences, warm air being drawn upwards through the chimney, especially with a fire burning. If there is no chimney, roof cowls are valuable, their action being supplemented by the force of the wind itself. Windows should be adequate, the usual allocation is said to be 5 square feet of window space for every 100 square feet of floor area. Mechanical ventilation is usually of propulsion type in large premises, but extraction ventilation is quite satisfactory providing that proper inlet for air is ensured, otherwise air is drawn in through cracks and under doors, producing draughts. It is also important to remember that windows should be closed in the near vicinity of the extractor, to prevent short circuiting of fresh air. Actual air conditioning does not seem to confer any additional benefit from the point of view of ventilation. The whole subject of ventilation is admirably dealt with in a detailed article by Bedford (1944).

(ii) Bactericidal mists. Much laboratory work has been published on the use of bactericidal mists, and has shown quite definitely that the dissemination of certain mists in the atmosphere of the laboratory in small concentration is sufficient to inhibit the growth of airborne pathogens. It remains to put this method to the test of a full-scale field trial from the clinical point of view.

The essential requisite of a bactericidal mist is that it should be bactericidal. In addition, it should be quite harmless, not unpleasant in the atmosphere, non-injurious to clothing or industrial products, non-irritant to the nares, non-corrosive to metals, cheap and easy to operate. Various agents have been tried, some of them unsatisfactory as they did not fulfil the above criteria; ozone, propylene glycol and resorcins have been used, and naturally the practicability of using a penicillin mist at once comes to mind. Of the first three substances, there is much to be said for resorcin. It is genuinely bactericidal in quite small quantities in the atmosphere, and can be very easily dispersed. This is possible by electrical heating of a small metal cup containing a solid cone of resorcin, from the surface of which a gentle wisp of the mist emanates into the room noiselessly, continuously and cheaply, without requiring any attention once the apparatus is connected with a power point. Propylene glycol is also efficacious, judging from its laboratory trials, and much work has been done with it in America and in this country.

Edward, Lush and Bourdillon (1943) have shown that rapid and effective sterilization of atmospheres containing atomized particles of influenza and vaccinia viruses, and probably also herpes simplex virus, can be achieved by ultra-violet radiation of wave-length 2537A. At least 99 per cent. and probably more of an aerosol of influenza virus was killed by exposure for 6 seconds at a distance of 2 cm. from either a Hanovia lamp or a G.E.C. Sterilamp. These results support the view that germicidal lamps are likely to be useful in reducing the infectivity of air contaminated with particles from persons suffering from virus infections of the respiratory tract.

Work done in the bacteriological laboratories at Guy's Hospital by F. A. Knott and the writer has demonstrated beyond a doubt the extraordinary effect of a penicillin mist in inhibiting the growth of organisms. Comparatively small quantities of the mist will inhibit growth of organisms at a considerable distance, for many hours after dispersal of the mist into the atmosphere. The passage of the mist through the apparatus does not appear to have the slightest effect upon the inhibitory action of the penicillin. It has to be remembered that the organisms are inhibited and not actually destroyed, and the addition of penicillinase will cause them to grow again. Moreover, at present the mist has to be delivered either by oxygen pressure from a cylinder, which is a costly business, or by means of an electric motor. The latter is cheap enough, and can be made noiseless by suitable enclosure. However, the solution of penicillin has to be replenished at intervals, so that the apparatus cannot be left to its own devices. It is also true that a great deal of penicillin is required to 'clear' a large room, when the apparatus is to be left running hour after hour, day after day, throughout the winter months, as would be required in any large-scale experiment. However, should large quantities of penicillin become available, it is interesting to visualize the possibility of using a mist of this kind on a controlled group of workers. It is also tempting to contemplate the possible effect of directing workers through a penicillin room on their arrival at work, bearing in mind the fact that exposure to the mist for only a few minutes appears to produce effective inhibition of bacterial organisms in the nasopharynx, an inhibition which persists for several hours.

Measures aimed at preventing the development of bronchitis in predisposed subjects who contract an upper respiratory infection must also be taken into consideration. These include both general treatment and the use of penicillin. It goes without saying that mitigation of the consequences of a head cold is to some extent a general health problem, inasmuch as the individual predisposed to bronchitis is more likely to suffer from an attack if, when he gets his cold, he is in poor physical shape, worn out by long working hours, and undernourished by the cumulative effect of some years of poor feeding. Should such a person catch a cold, as sooner or later he naturally will, then special care must be taken. He should stay at home from work, and go to bed, where he should remain for at least two days in a warm even temperature, or until his cold is better.

Various measures have been in vogue from time to time for their supposed value in limiting respiratory infection. Of these general sunlight is still popular but, although comforting, there is no real evidence that its use is successful in raising resistance to any appreciable extent. Cod liver oil has been given for the same purpose and, although here too, assessment of its value is a matter of great difficulty, it has at least stood the

test of time, and the clinical impression of many very experienced physicians is that cod liver oil has some value in building up resistance to winter colds.

Different kinds of vaccines have been, and still are, in use as prophylactics. A stock anti-catarrh vaccine is commonly advised, but large-scale experiments carefully controlled have not shown any appreciable protection in the treated groups. It does seem, however, that the autogenous vaccine is of some value as, although it cannot be said to prevent upper respiratory infection in bronchitis, its use appears to ameliorate the severity of the symptoms of any resultant bronchitic attack, and very frequently the attacks themselves are fewer in number.

A considerable amount of investigation has now been made in the use of penicillin by spray and inhalation through the respiratory passages. Various facts emerge. Of these perhaps the most important is the observation that penicillin can be absorbed through the respiratory mucosa in sufficient amount to produce a therapeutic level in the blood, and of course very considerable quantities will then appear in the urine. Moreover, the use of sprays on the nasopharynx has a definite therapeutic value in limiting the length of infectivity of streptococcal and staphylococcal conditions there. It seems reasonable to hope that inhalations of penicillin at the beginning of a head cold may serve to prevent the onset of bronchitis in those individuals in which this is the usual sequence of events, and certainly preliminary clinical results obtained by the writer tend to strengthen this impression.

From these results it appears that no benefit is likely to follow the use of penicillin inhalations in the treatment of longstanding respiratory disease, such as established chronic bronchitis, or bronchiectasis, in which the extensive peribronchial fibrosis and poor air entry together contribute formidable obstacles to the production of any permanent improvement. In these conditions little if any clinical improvement has been noted after even a prolonged administration of penicillin over a period of some weeks, with an hour or more inhalation at a daily session, and a total dosage exceeding 3 million units. The more acute catarrhal infections, on the other hand, do appear to be a much more promising prospect, as already indicated. Although it is, of course, impossible to reach any firm conclusion based on the study of a small group of cases, it would appear justifiable to treat any head cold or upper respiratory infection in a bronchitic subject with a prophylactic course of penicillin by inhalation.

The technique of penicillin inhalation is not difficult. The apparatus used by Mutch and Rewell (1945) in their experiments in this mode of administration was an inhaler originally designed for giving adrenaline mists to asthmatics, and modified for this special purpose with specially wide-bore tubing and a closely fitting face-mask. The actual inhalation is not unpleasant at all. It is true that there is an odour somewhat reminiscent of the farmyard, but patients do not find it intolerable, and it is reassuring to know that the yellow staining of the face and moustache washes off quite easily. For the purpose of a large-scale experiment, a less extravagant apparatus is desirable, and in fact it is not difficult to produce an excellent mist with an electrically driven air current. Up to the present it has not been possible to nebulise a fine enough mist with any form of hand spray; the particles produced are much larger, and are deposited in considerable quantity in the naso-pharynx, where they are swallowed and do not reach the bronchial tree itself.

Treatment of the established case of Chronic Bronchitis

Apart from advice on general health, and the avoidance of the occupational precipitants noted earlier in this article, the duty of the physician towards the patient with real chronic bronchitis lies chiefly in the treatment of the obstinate cough, the accompanying bronchospasm, and the progressive pulmonary emphysema.

Some observers in the past have stated that no expectorant mixture is of any value at all, and have sought to prove that after administration of expectorants there is no increase in the amount of sputum produced. Whatever may be the laboratory evidence, however, all bronchitic sufferers will testify that the act of expectoration is facilitated by certain mixtures, and the distress of coughing much relieved. Not all expectorants are of equal value in this respect, and it is the strong conviction of the writer that the two of greatest worth are potassium iodide and ammonium carbonate. The former especially has a notable effect on the sputum, liquifying it and relieving bronchospasm to some extent. It should be given indefinitely in small doses throughout the winter months to these patients. To be of any use, ammonium carbonate, although unpleasant, must be given in large doses. In addition to the above drugs, the Brompton hot water mixture is often a great help in the mornings, and consists of sodium chloride, sodium bicarbonate, spirit of chloroform and aniseed water, and is given in an ounce of hot water. One other small point is the inadvisability of the common routine addition of stramonium or belladonna to these mixtures, because of their action in drying up secretions.

For the treatment of emphysema it is permissible to emphasize the surprising success of physiotherapy in some of the most unfavourable cases. Relaxation, deflation of the lungs and added mobility to the thorax, together with improved expiratory breathing power, and an increase in the vital capacity itself, can all be achieved by patient tuition in a well organized clinic, providing the subject is allowed the necessary time to attend, for it is usually many months before the maximum benefit is attained. The use of a belt to hold in the abdomen and elevate the diaphragm, thus diminishing the amount of residual air in the lungs, has been advised and is theoretically sound, but patients themselves dislike the sense of constriction round the waist and are loath to adopt the method.

Ephedrine is of great value, even in the absence of any obvious bronchospasm which, however, always exists to a certain extent in these cases. It should be given in full doses at regular intervals for an indefinite period. Some patients will complain of its side effects, such as palpitation, and it may then be found that administration as a mixture instead of as the usual tablet overcomes this drawback. It is also worth while bearing in mind that elderly bronchitics may complain of the difficulty in passing water after taking ephedrine.

Conclusion

The writer has been much impressed by the constancy of the infective factor in the great majority of cases of recurrent bronchitis. As it appears impossible at the present time to do more than ameliorate the symptoms of the chronic case, it is felt that attention should be directed towards the prevention of the preceding recurrent infective episodes. There seems to be sufficient laboratory evidence to justify the experimental use of bactericidal mists and inhalations on patients themselves.

It is suggested that it would be profitable to undertake a field trial of these methods in industry, or wherever there are large numbers of persons under continued supervision, especially where they have properly documented past medical histories.

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THE INCIDENCE OF SIDEROSIS IN IRON TURNERS AND GRINDERS

BY

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Doig and McLaughlin (1936) drew attention to the x-ray changes in the lungs of electric arc welders, and suggested that, amongst other possibilities, the iron oxide particles inhaled from the welding fume might be opaque to x-rays and produce the picture without the presence of pulmonary fibrosis, or congestion. They were unable to confirm this theory with necropsy material but Enzer and Sander (1938) produced proof of its validity by histological studies of the lungs of a welder whose x-ray chest film had shown similar changes. Since then the picture has been observed by many more workers, including Britten and Walsh (1940), Harrold, Meek and McCord (1940), Sander (1944) and Groh (1944). Pendergrass and Leopold (1945) recorded an account of four cases of siderosis or 'benign pneumoconiosis' occurring among 50 grinders of bearings made of chrome vanadium and chrome molybdenum tool steel containing 98 per cent. iron, about 2 per cent. alloy and not more than 0·2 per cent. silica. Artificial abrasive wheels composed of bakelite, silicon carbide (carborundum) and aluminium oxide (aloxite) had been used exclusively in the works for the previous 17 years. The four workers who showed radiological changes had been employed for twelve, thirteen, fourteen and seventeen years respectively. The dust to which they were exposed had a concentration of 4·9 million particles per cubic foot of air. 96·5 per cent. were less than 5μ in size, and 99·5 per cent. less than 10μ. The average percentage of silica, as quartz, in the dust was 0·43. McLaughlin, Grout, Barrie and Harding (1945) further showed that a similar x-ray picture can be produced in workers using iron-oxide (rouge) to polish silver; one of the workers died and they showed that, after 40 years, there was no fibrosis present in his lungs. These authors reviewed the literature of siderosis which was first described by Zenker in 1866, but he used the term with the implication that iron and iron oxide dust causes fibrosis in the lungs. The industrial implications of the condition, however,

TABLE 1

DETAILS OF PROCESSES

Shop	Light machine Gallery	V.S.O.	No. 2 Turnery	No. 1 Turnery Grinding section	No. 1 Turnery Heavy bay
Type of machine	Turret lathes	Hydromatic milling machines	Centre lathes Turret lathes	Internal grinding	Planers, plano mills, borers, and milling machines
Metal	Cast iron	Mainly cast iron	Cast iron	Cast iron	Cast iron and steel
Speeds	37 to 312 r.p.m.	68 Spindle speed	Various	1,500 r.p.m.	Various
State of castings	Castings turned completely clean. In almost all cases fettled	Fettled before milling	Turned clean	Castings fettled or shotblasted	Fettled
Wet or dry ..	Dry	Dry	Dry	Operation completed with dust extractors	Cast iron—dry Steel—water
Other dusty processes ..	None	None	None	None	None

FIG. 1.—Male. Aged 43. Iron turner for 18½ years.

were not seriously considered in England until 1923, when Collis pointed out that skiagrams of the chests of haematite miners showed the presence of shadows suggestive of fibrosis of the lungs, though 'the opacity of oxide of iron to x-rays rather detracts from the significance of these findings.' Harding (1945) confirmed this by producing radiographic stippling or fine nodulation in the lungs of rats by intratracheal injection of rouge. No fibrosis was detectable in these lungs as a result of the presence of iron oxide for some eight months. The fact, however, that fibrosis is not produced does not justify the presence of large quantities of iron or iron oxide in the atmosphere, because it is unlikely that the lung can become a physiological dust trap and retain completely its elasticity and function.

In continuation of the work described above it was thought desirable to study the radiological state of the lungs of men and women who had been exposed to dust in iron turneries. In the factory chosen for the investigation the work of turning and grinding cast iron was carried on in five shops. Details of the machines and turning speeds are given in Table 1. It will be seen that turret lathes, milling machines, centre lathes, planers and borers were used as well as grinding machines. The speeds of turning varied from 37 to 1500 revolutions per minute, turning at high speeds being much dustier than at low speeds. Before the castings came to the turning shops adherent sand had been removed by shotblasting and by fettling with pneumatic hammers. Some of the castings had been weathered and had a deposit of rust on them. The turning was done dry in the case of cast iron, but sometimes

turning of steel castings was done in these shops, and then liquid coolants were applied to the cutting tools of the machines. Grinding of cast iron by abrasive wheels was performed under exhaust ventilation. There were no other dusty processes in the shops.

A sample of dust obtained from a rafter above the 'Wider Top Cutter' had the following composition:

	Per cent.
Combustible matter (carbon, oil, bits of fibre)	12·2
Total silica	5·4
Silicate residue	1·3
Metallic iron	37·1
Iron oxide (calculated as Fe_2O_3)	23·3
HCl. soluble matter other than iron	20·7
	100

It is realised that rafter dust does not necessarily represent the dust inhaled by the workers, but it gives a good indication of the type of dust present in the general atmosphere of the shop.

There were employed in the turneries 50 women and 161 men, of whom 33 women and 138 men volunteered to be x-rayed. Their ages were noted, as well as the length of time they had worked in turneries. The radiographs were studied and occupational histories were obtained from all those who showed any abnormality, in order to be certain that they had not had previous employment in a dusty occupation. They were also asked if they had had any symptoms such as shortness of breath, cough or wheeze.

In no instance, was nodulation seen in the x-ray films, and for the purpose of this report the cases showing reticulation were only regarded as abnormal if the changes were agreed to by three of us (M. H. J.,

FIG. 2.—Male. Aged 53. Iron turner for 30 years.

FIG. 3.—Male. Aged 46. Iron turner 29 years. Contact print of right lower lobe.

A. I. G. McL., and K. M. A. P.) studying the radiographs independently. The reticulation found was never gross, and was of a peculiar, fine character, the 'stippling' or individual components of the reticulation being so fine and so closely sown as to produce a ground glass appearance such as is seen in cases of asbestosis. In what may be called early cases of siderosis the ground glass effect appears first in the lower lung fields, possibly because iron dust is heavy (figs. 1, 2, and 3). The changes were present in 15 cases; the highest incidence was among workers who had performed this work for more than 20 years—5 out of 34. It will be seen (Table 3) that the changes began to appear in the x-ray films in workers who had been exposed to the inhalation of the dust for six to ten years, but that none of the seven workers who had worked in the turneries for 11 to 15 years, and only one out of 11 of those who had worked for 16 to 20 years showed the changes. The reason for this is obscure, though individual variations in dust-eliminating mechanism of the lung may partly account for the discrepancy. The incidence by age (Table 2) was 5 out of 44 in the thirties, 5 out of 40 in the forties, and 4 out of 23 in the fifties. One man had previously been exposed to silicosis risk; for six years before starting work in the turnery, he had been a fettler in an iron foundry. There were no positive cases in the 15 men over 60 years of age. In the interpretation of the chest films of elderly people it is necessary to remember that changes are often present in normal individuals due to chronic bronchitis and the degenerate process of ageing, and it is possible an over conservative interpretation was made in this group. It is probable, therefore, that the changes are caused by the iron dust inhaled at work, and they are clearly not primarily related to age, but rather to length of exposure. No symptoms appeared to be related to the changes; of the 15 men only one complained of shortness of breath, and none of wheeze, though six complained of cough. The cough could not necessarily be attributed to the dust; the symptom is extremely common in the middle-aged male population; it may equally well be attributed to the present-day heavy consumption of tobacco. None of the women, all of whom were young, and none of whom had been working in the factory longer than five years, showed abnormal x-ray changes. The full details are given in Table 2 by ages, and Table 3 by length of exposure.

In a few cases x-ray evidence of other diseases in the lung was found. Arrested pulmonary tuberculosis was found in five cases, but in no instance was there any clinical evidence to suggest recent activity, and no active cases were observed. Emphysema was present in 8 cases, one of which showed a large bulla at the left apex.

Differential diagnosis of the various types of dust disease is not possible on x-ray evidence alone. The radiologist cannot distinguish between

TABLE 2

INCIDENCE OF SIDEROSIS, DISTRIBUTION BY AGE

FEMALES

Turnery	No. of workers	Age				
		29 and under	30–39	40–49	50–59	60 and over
No. 1	17	14	3	—	—	—
No. 2	16	15	1	—	—	—
Total ..	33	29	4	—	—	—
MALES						
No. 1	44	8	15 (1)*	13 (2)	5	3
No. 2	94	12 (1)	25 (4)	27 (3)	18 (4)	12
Total ..	138	20 (1)	40 (5)	40 (5)	23 (4)	15
Combined Total ..	171	49 (1)	44 (5)	40 (5)	23 (4)	15

* Previously fettling iron castings for six years.

Figures in brackets indicate number showing x-ray reticulation.

TABLE 3

INCIDENCE OF SIDEROSIS, DISTRIBUTION BY LENGTH OF EXPOSURE

FEMALES

Turnery	No. of workers	Length of exposure in years					
		Under 2	3–5	6–10	11–15	16–20	Over 20
No. 1	17	17	—	—	—	—	—
No. 2	16	13	3	—	—	—	—
Total ..	33	30	3	—	—	—	—
MALES							
No. 1	44	—	16	14 (1)*	4	2	8 (2)
No. 2	94	3	23 (3)	30 (5)	3	9 (1)	26 (3)
Total ..	138	3	39 (3)	44 (6)	7	11 (1)	34 (5)
Combined Total ..	171	33	42 (3)	44 (6)	7	11 (1)	34 (5)

* Previously fettling iron castings for six years.

Figures in brackets indicate number showing x-ray reticulation.

the x-ray nodulation associated with the fibrosis produced by silica and that caused by opaque substances such as iron oxide or barium (Arrigoni 1933; Pendergrass 1938; Preti and Talini 1939). The symptoms produced by siderosis are negligible, whereas the shortness of breath caused by silicosis may be disabling. It is important, therefore, to differentiate between the two conditions, and this can only be done by means of a detailed occupational history and a first-hand knowledge of the environmental conditions to which the worker is exposed.

Summary

One hundred and seventy-one individuals (33 female, 138 male) exposed to iron dust in iron turneries were radiographed in order to gauge the incidence of changes in their lungs due to iron oxide. These changes, in the form of reticulation, were present in 15 instances. In 5 cases the man had been working in this occupation for more than 20 years. The changes were not gross, symptoms were few, only one complaining of shortness of breath though six said they had some cough.

The dust obtained from rafter samples contained 37·1 per cent. metallic iron, 23·3 per cent. iron oxide, while the total silica content was 5·4 per cent. and the silicate residue 1·3 per cent.

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OCCUPATIONAL DERMATITIS

BY

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Liverpool

Dermatitis is any inflammation characterized by the classical signs of redness, swelling, heat and pain. It is a non-specific reaction of the skin to an irritant and cannot be considered an entity. Occupational dermatitis is such a reaction produced by, or aggravated by, an irritant encountered during the course of employment. At the present time the word dermatitis is almost synonymous with occupational skin disease and the word has unfortunately become of more legal than medical significance. In view of the diversity of industrial processes and the wide range of substances which will produce irritation of the skin in certain people with average skins, it is not surprising that, with few exceptions, the eruption is not characteristic. Pain is not usually a dominant feature and is more commonly replaced by itching. The onset is occasionally sudden, but more often insidious. The appearance of the skin eruption is proportional to the severity of the reaction produced by the causative agent and the stage at which the eruption is seen. These two factors are more important than the nature of the causative agent in considering the appearance of the eruption, although some agents produce a constant and characteristic lesion. The usual sequence of events is erythema, oedema, papules, vesicles, or more rarely bullae which on rupture exude serum. The serum dries and produces crusts. When oedema of the prickle cells occurs the granular and horny layers are imperfectly formed and adhere together to produce scales. The appearance of the eruption is frequently complicated by subsequent scratching and infection.

Pathological changes in the skin accompany the signs. The blood vessels of the corium are dilated and there is exudation of lymph and infiltration of small round cells. The cells of the epidermis are swollen and separated by fluid which produces spaces forming visible vesicles. The disease produced is the reaction of the skin to an irritant which may reach the skin from without or within. The eruption arising from the action of an external irritant is the more frequent, but many substances are known to cause skin rashes when taken internally, and possibly eczema is a type of dermatitis produced by an irritant resulting from metabolic processes.

As has been shown by Graham-Little (1935), the occurrence of occupational dermatitis is of great social importance. The reports of the Chief Inspector of Factories show that 8926 cases of dermatitis were voluntarily notified in 1943, compared with 8802 in 1942, 4744 in 1940, 2952 in 1939 and 2195 in 1938. These cases may be considered a fair sample of those which occur annually in industry, but no complete figures are available. The American Public Health Service made studies among more than 100,000 workers in various factories and found that in the period of a year about 1 per cent. of all workers in those factories were affected with occupational dermatitis, though burns, splashes of acid or alkalis or pyogenic infections of occupational skin wounds were not included (Schwartz, 1943a). The incidence of the disease varies widely in different industries, but many cases of industrial dermatitis occur among Engineers, Dyers, Metal Workers, Textile Workers, Chemical Workers, Painters and Polishers.

The following figures are representative of the incidence of skin disease in the chemical industry. The cases were observed during the six years 1935–41, when every man received a complete physical examination.

Table 1 records the number of employees engaged in the industry, in age groups, and Table 2 shows the type of skin disease discovered.

TABLE 1

EMPLOYEES EXAMINED

Number examined	Age
18	15
247	15–20
331	20–25
336	25–30
382	30–35
353	35–40
318	40–45
302	45–50
276	50–55
200	55–60
203	60–65
Total 2966	

TABLE 2

TYPE OF SKIN DISEASE	
Acne, including occupational acne	169
Occupational dermatitis excluding acne	75
Eczema	21
Varicose eczema	10
Psoriasis	9
Impetigo	6
Seborrhoea	44
Acne Rosacea	3
Fungus Infections	4
Scabei	1
Furunculosis	10
Urticaria	5
Alopecia	1
Ichthyosis	1
Total	359

Practically every occupation can cause skin irritation and estimates of the relative prevalence of occupational compared with other skin affections vary between 2 and 20 per cent., 6 per cent. being a conservative estimate (White, 1928). Innumerable substances produce dermatitis but among the common ones are alkalis, acids, oil, degreasing agents and sugar, dough, salts of chromic acid, dyes, dinitrochlorbenzene, formaldehyde, tar and naphtha; soapy water, petroleum products, dyes and intermediates, paints, cleansing preparations, hair tonics and lime, chrysanthemum, pyrethrum, geranium and many types of bulb. Paraphenylenediamine is a common constituent of dyes for fur and hair, and is a powerful sensitizing agent. Many drugs applied locally will produce skin reactions, and the use of atropine is sometimes followed by dermatitis of the face. Cleansers such as alkalis, bleach, solvents and abrasives are often the cause of occupational dermatitis rather than the contacts which they are called upon to remove and a factor of importance in considering the prevention of the disease. A comprehensive list of causes has, however, been compiled by Schwartz (1943b); and O'Donovan (1932) stressed the importance of dermatitis arising in the home, and suggested the following classification of skin diseases caused by agents acting externally:

1. Mechanical or physical agents.
2. Plants and their products.
3. Living agents.
4. Chemicals.
   - (*a*) Inorganic acids and salts.
   - (*b*) Hydrocarbons, oils, tar, pitch, anthracene.
   - (*c*) Dye products.

Most cases of occupational dermatitis may, however, be arranged under headings describing the main action of the causative agent:

1. Mechanical. Inert dusts, e.g. sand, pumice, silica.
2. Macerative. Water and alkalis.
3. Fat solvent. Petrol, paraffin, benzene.
4. Chemically irritative. Acids and alkalis, dyes, mineral oils.

Types of Occupational Dermatitis

A. Primary Skin Irritants

These produce clinical irritation of the skin in the majority of persons who have not been previously sensitized to the causative agent. They may give rise to non-characteristic and characteristic eruptions. The former comprise the majority of cases. The lesions may be confused with a number of infective and constitutional skin diseases, but in the troublesome type of case the differential diagnosis is between constitutional eczema and occupational dermatitis, between a skin eruption produced by internal and one by external irritation; while the latter are a group of less common but specific types of lesion, such as the rashes produced by oil and the acne produced by chlorinated hydrocarbons.

B. Secondary Skin Irritants

These produce reactions only in the skin of persons who are hypersensitive, the sensitization being acquired by contact with the causative agent. This type of dermatitis is less frequent than that produced by primary skin irritants, but is often more severe, and the disease may be protracted and subject to relapse on recontact.

Over a period of ten years, 92 cases of non-characteristic occupational skin disease have occurred in a chemical industry, which employs, on the average, 2000 people. Of these 67 were caused by primary skin irritants, and 25 by secondary skin irritants. Tables 1 and 2 record the majority of the cases and also the incidence of all skin diseases during a six-year period. Cases of occupational dermatitis occurring subsequent to this review are included in the following classification. The progress of many of the cases recorded has been observed over a period of several years.

A. Primary Skin Irritants

1. *Non-characteristic Eruptions.* The signs of inflammation are evident in the area exposed to the causative agent. In its mildest form the disease is a localized erythema and if the case is immediately removed from contact with the irritant and simple remedies applied an early and permanent cure is to be anticipated. Early removal from contact is a measure of outstanding importance in order to prevent widespread disease which may result in skin sensitivity to many agents. In the mild type of disease the worker usually becomes immune, but with severe and extensive lesions no immunity is to be expected. In severe cases the lesions are eczematous in type, the features of which are described later.

It is possible to distinguish three groups of cases which arise from primary skin irritants.

(*a*) The group of cases described above which can usually be readily explained. Forty-one of the 67 cases due to primary skin irritants belonged to this group.

FIG. 1.—Acne resulting from exposure to chlorinated naphthalenes.

FIG. 2.—Acne arising from exposure to chlorinated naphthalenes.

FIG. 3.—Acute dermatitis arising from exposure to dinitrochlorbenzene.

(*b*) A group of cases arising in employees who have been engaged on the same work for many years without any evidence of skin disease. Eighteen of the 67 cases belonged to this group. They developed a serious, extensive and protracted disease, never became immune and usually developed, in addition, a sensitivity to many of the materials used in daily life. Mumford (1934) assumes that the production of some substance derived from damaged skin has lowered its resistance to any irritant; but Hall (1939) concluded that the skin undergoes changes in structure and composition at different periods of life and cites this type of occupational dermatitis and infantile eczema as examples. One such case occurred in a shift worker aged 55 years who was engaged in packing bleach for 41 years (1892–1933). He first noticed a few pimples on the front of the left elbow in March 1933, which quickly spread to other areas. Papules and weeping patches developed on the backs of the hands, forearms and arms, and slight erythema on the neck and inner sides of the thighs. He was obliged to cease work. The condition improved very slowly until 29.6.34, when he became free from any eruption, but relapsed after a few days and, in spite of hospital treatment, his skin could not be rendered clear until 2.12.35. Within a few days of his discharge the hands were again affected.

(*c*) A group of intractable cases occurring in related workers having some features of sensitization dermatitis. Several members of the same family were affected and among the 67 cases already quoted, 8 belonged to this group and there were family relationships thus—two brothers and one nephew; two brothers, father and daughter; while one case had an uncle affected with occupational dermatitis and a brother with psoriasis. The cases as a group are similar and present distinctive features. The initial skin lesions are out of all proportion in extent and severity to the known properties of the causative agent. The reaction is eczematous in type and occurs in areas not exposed to direct irritant action. Relapses are frequent and imply an inherent instability of the skin in which simple external factors (soap, friction, perspiration) induce excessive local response. The history of such cases supports the view that there is little possibility of returning them to regular normal work and if a family history of skin disease is discovered before employment they should be excluded from contact with any known skin irritant.

2. *Characteristic eruptions* are produced by a number of agents and include chromic acid burns, tar warts and epitheliomata.

Oil rashes are manifested by blackheads and pustular eruptions which occur on parts of the body contaminated by oils, particularly after slight injuries such as those caused by metal filings. This form of dermatitis is produced by blocking and irritation of the skin follicles followed by septic infection by organisms generally derived from the skin surface. Factory Dept. (1932). Whitwell (1943, 1945), however, has shown that all cutting agents are not equally harmful to the skin, and chemical irritation may be the primary cause and not mechanical blocking of the skin follicles.

Acne. The occurrence of acne in industry has been noted for many years and has usually been described under the name of 'chlor acne.' This is a misnomer, inasmuch as exposure to chlorine, or to many compounds containing chlorine, does not give rise to the disease. The majority of workers exposed to the fumes of chlorinated naphthalenes and chlorinated diphenyl will develop acne if the exposure is continued for any length of time. The lesions are seen earliest and most commonly on the face, commencing external to and below the eye to the malar prominence and spreading over the cheeks. Numerous comedones are present behind the ears. Few are present on the forehead and the nose is characteristically spared, as is the mid-line of the face generally. In severe cases the lesions are found on the neck, chest, upper abdomen, arms and extensor aspects of the forearms, particularly about the elbows. The most characteristic feature is the enormous number of very fine blackheads around the eye, and this observation associated with the distribution of the eruption and the absence of noticeable greasiness, distinguishes the eruption from acne vulgaris (Jones, 1941).

B. Secondary Skin Irritants (Sensitization dermatitis)

Certain types of skin disease are related to hypersensitivity and allergy. Allergy is defined by Bray (1931) as 'exaggerated susceptibility to various foreign substances or physical agents that are harmless to the great majority of persons.' The term should be restricted to designate reactions which occur after a lapse of time following the initial lesion and which are often severe and produced by slight contact with the original irritant. MacKenna (1934), reviewing some of the knowledge concerning anaphylaxis and allergy which has accrued from numerous exact and scientific experiments, stated that an anaphylactic reaction can be obtained in a majority of animals and in man, but a person is not easily made allergic. It seems to be agreed that the percentage of allergic persons in the population is between 0·5 and 1.

Only a small percentage of cases of occupational dermatitis are inherently allergic in type, but many more occur among the users of finished products. In these cases the patient becomes sensitized to a particular causative factor and will relapse immediately if contact is again established even with minimal exposure. Subsequent attacks are

likely to be more extensive and intractable. There appears to be a danger that, if a severe attack is experienced the skin may become so affected that contact with any irritant will cause a relapse. Irritants which give rise to sensitivity include many dyes and intermediates, and Ingram (1942) states that the local application of sulphonamides may produce sensitization, while Tate (1944) records epidermal sensitization with eczematous reaction produced by local application of sulphonamides which may be so intense as to preclude their subsequent administration in effective doses for other diseases. Sensitization may follow the use of any of the sulphonamides.

The type of reaction produced is often characterized by the sudden onset of erythema, oedema and weeping dermatitis associated with severe irritation —often widespread and involving the face and other exposed parts. In atopic sensitization reactions occur away from exposed areas.

It has been shown that by cutaneous administration of simple chemical compounds, such as 2 : 4 dinitrochlorbenzene, in small quantities, a typical incitant of contact dermatitis in man, it is possible to induce true anaphylactic sensitization in guinea-pigs, demonstrable by the intravenous injection of protein conjugates. Landsteiner and Chase (1937) suggest that this is strong evidence in favour of the formation of antigenic conjugates following application of substances of simple chemical constitution. Sensitivity to dinitrochlorbenzene may be extreme and four individuals were found to develop severe reactions from a 1 in 1,000,000 solution (Jones, 1934). The following history illustrates a case of this type:

H. T. Born 12.8.82, a chemical process labourer, developed erythema and oedema on the forearms, arms, neck and face on 22.1.16 from contact with dinitrochlorbenzene dust. He recovered in 3 weeks but relapsed immediately after contact with the same material and was absent for 6 months during the second attack and has not been free from symptoms since. On 14.12.33 he was tested with dinitrochlorbenzene in alcohol and reacted violently to one drop of 1 in 1,000,000 solution.

The areas affected on 17.5.34 were the backs of the hands and fingers which were red and cracked and the flexor aspects of the forearms which showed erythema and papules. Repeated attacks of erythema, papules and weeping occur on the hands, face, forearms and thighs without obvious cause.

Diagnosis

The diagnosis of occupational dermatitis may be difficult as in most cases the changes in the skin are no different from those that occur in dermatitis or eczema of non-contact origin and there is no specific type of reaction corresponding to any particular irritant. The early diagnosis of the condition is of the highest importance to treatment. The removal of the person from contact prevents the development of the intractable type of case which develops sensitivity to a multiplicity of agents. Schwartz (1940) stresses that the two most important factors in the diagnosis of occupational dermatitis are the history and the site of the eruption. However, the diagnosis must mainly depend on a full consideration of the mode of development of the eruption, its distribution in relation to areas exposed to irritation, the exact nature of the work and irritants handled, and the materials used to cleanse the hands, together with the patient's previous skin history (Thomas, 1939). Horner (1934) stresses the frequent fallacious description of the work performed by the patient which is no guide to the actual cause. An attempt should be made to establish the following points in each case:

1. Whether an occupational causal factor has been established at some previous time.
2. Contact with an agent known to produce similar changes in the skin has occurred.
3. The time relationship between exposure to the agent and onset of the dermatitis is correct for the particular agent and abnormality of the skin.
4. The site of onset of the skin disease is the site of maximum exposure.
5. The lesions present are consistent with those known to have followed the reported exposure or trauma.
6. The individual is employed in an occupation in which similar cases have previously occurred.
7. No exposure outside occupation can be implicated.

If dermatitis is established the following confirmation is important:

(1) Evidence of previous attacks after exposure followed by improvement after cessation of exposure. Many cases improve at weekends or after holidays.
(2) Patch tests corroborate the history and clinical findings. (Foerster, *et al.*, 1942, 1943.)

Site of Eruption

The site of the disease is related to the physical nature of the irritant and also to chemical factors which determine its class, in addition to the mechanical effects of the process being worked. Dermatitis produced by direct contact appears commonly on the backs of the hands and fingers, and flexor aspects of the forearms; while the palms usually escape. Liquids may splash or spray and the face may consequently become affected. Dust spreads widely and causes an eruption on the neck, axillae, perineum, genitals, feet and ankles, and belt area, i.e. the sites of friction. The skin is comparatively thin over the inner surface of the forearm and the upper arm, and these areas are often affected. True industrial dermatitis of the eczematous type always begins on the parts most exposed to the action of the irritant causing it, and in the majority of cases these are the hands and arms. In severe cases and where sensitization occurs the rash may spread to other parts of the cutaneous surface remote and well protected from the direct action of the irritant. The eruption may persist in these parts, as well as on the exposed parts, for an indefinite period with

FIG. 4.—Acute dermatitis arising from exposure to dinitrochlorbenzene.

FIG. 5.—Chronic dermatitis resulting from exposure to dinitrochlorbenzene.

remissions and relapses. In fact, it behaves like a constitutional eczema, from which it can only be distinguished by the history of its original incidence (Haldin-Davis, 1939). Some consider that a dermatitis due to work cannot persist for a long period after the removal of the alleged irritant, but others hold that the human skin which has once been attacked by occupational dermatitis may acquire a habit of breaking down, and becoming sensitive to agents and influences which were previously harmless.

Appearance of Eruption

The appearance of the lesions is proportional to its stage and severity; and various types have already been considered. In a majority of cases the eruption is not characteristic and difficulty arises in those cases which resemble constitutional eczema and where there is no obvious contact between the skin and an irritant. Stokes (1930) has defined eczema as a dermatitis in which the constitutional outweighs the exciting cause, but the word is used in different senses and by it the majority of English dermatologists mean an inflammation of the skin in which the epidermis is most conspicuously involved showing the histological changes known as spongiosis. Clinically tiny vesicles are formed. It is clear that eczema must have a large variety of causes and it is unsound to call the disease ' eczema ' when the noxa is unknown and exclude from this title identical manifestations produced by a known noxa. The connection between eczema and ' dermatitis,' in which participation of the papillary body and cutis is more pronounced, is very close and clinically it makes little difference whether the epidermis or dermis is the seat of primary damage, for it is impossible for a reaction to take place in either without secondarily influencing the other. The difference in the site of maximum reaction may be largely a matter of intensity and exogenous eczema-producing substances, if in too weak concentration or used for too short a time, produce an erythema. The proportion of cases of eczema attributed to external noxae varies with the energy expended in searching for them (Goldsmith, 1936). MacCormac (1937) points out that the age of incidence of idiopathic eczema and industrial dermatitis, particularly in males, have a very similar curve. Probably eczema and dermatitis are one and the same reaction and are the same condition pathologically, but not aetiologically; it is, therefore, essential to distinguish between eruptions caused by exogenous irritants and those produced by internal constitutional causes. Familial diseases such as infantile eczema, asthma, hay fever, urticaria must be recognized and considered in arriving at a diagnosis. Hallam (1938) separates dermatitis into traumatic, atopic (which includes infantile eczema and sequelae) and sensitization (often referred to as eczema). He considers that infantile eczema and prurigo should be regarded as an entity and distinguished from other eczemas. Atopy also includes predisposition to particular types of allergy such as asthma and hay fever.

It is impossible, therefore, to establish a diagnosis of occupational dermatitis from the appearance of the eruption in a high proportion of cases, and the following principles should be fulfilled. The aforementioned criteria should be satisfied; the causative agent should be identified if possible and classified skin diseases must be excluded before the diagnosis is established.

Identification of Irritant

The isolation of the responsible irritant may be difficult. In spite of the complexities and variations of individual response and the immense number of potential irritants, the clinical picture varies but little. The exact cause cannot be recognized from the symptoms alone; they consist of erythematous, irritable, diffuse patches—of various sizes, round or oval in shape—and moist, dry, fissured, or scaling according to the stage in which the patient presents himself and not infrequently according to what has been applied for relief (Semon, 1939).

Many different substances cause dermatitis and, if suspected, can be applied to the skin in the form of a patch test. An adequate review of this method of diagnosis calling attention to some of the difficulties and necessary precautions is recorded by Levin (1942). A careful technique is necessary. Different parts of the body vary in sensitivity and tests should be applied as near as possible to the site of inflammation. Patch tests are an important aid to diagnosis, but responsible for many errors when relied on for diagnosis, and when used to detect primary irritants. Their value lies in deciding the cause of hypersensitivity dermatitis and mistakes may be avoided by a careful history. Clinical experience suggests that states of hypersensitivity to a chemical irritant which in full strength is a primary irritant will always give a positive reaction to 1 in 10 dilution (Osborne and Hallett, 1942). The conclusion arrived at is that these tests are sometimes of corroborative but rarely of primary diagnostic value in this connection, and, furthermore, that reactions are not specific (Rep. Chief Insp. Fact., 1936). Their practical value is considered doubtful by Silcock (1944).

Differential Diagnosis

A very large number of skin diseases are outside the influence of occupation and must be excluded. So far as the dermatologist knows, at present, these are produced by internal and quite undetermined causes. Such diseases often mimic or complicate the eruptions due to occupation (White, 1932).

In considering differential diagnosis it is instructive to note the incidence of skin disease in the Out Patients Department of hospitals and skin clinics. There are about 300 skin diseases recorded and one-third of these are fairly common, 20 are common in general practice. Ninety-five per cent. of cases of skin disease seen at Out Patients Depart-

FIG. 6.—Chronic dermatitis resulting from exposure to dinitrochlorbenzene.

FIG. 7.—Chronic dermatitis resulting from exposure to dinitrochlorbenzene.

ments are inflammatory and either due to some external cause, and can be considered as manifestations of occupational dermatitis or eczema, or to septic infection such as impetigo or furunculosis, or to various permutations and combinations of these conditions. The remaining 5 per cent. consist of scabies, alopecia areata, ulcers of the legs, tinea infections and an occasional lichen planus or pityriasis rosea (Haldin-Davis, 1935). Semon (1939) after thirty years' experience in Out Patient Clinics states that the incidence of industrial dermatitis averages about 10 per cent. of all cases treated. Roxburgh (1944) is of the opinion that thirty common skin diseases are mistaken for industrial dermatitis. Among the chief diseases to be considered the following are important:

Scabies.	Syphilis.
Lichen planus.	Pompholyx.
Varicose dermatitis.	Impetigo.
Mycotic dermatitis.	Pityriasis rosea.
Seborrheic dermatitis.	Erythema multiforme.
Pediculosis.	Drug eruptions.
Psoriasis.	Dermatitis herpetiformis.
Urticaria.	Ichthyosis.
Flexural eczema.	Lupus erythematosus.
Folliculitis (Sycosis).	Intertrigo.
Ery. Scarlatiniforme.	Prurigo Aestivale.

In differentiating them it is important to examine the patient stripped. Fungus infections of the hands are not very common and the agent should be demonstrated to establish the diagnosis. The feet are commonly affected and can often be diagnosed on clinical grounds. Epidermophytid eruptions on the hands are common and may be mistaken for occupational dermatitis. Pompholyx is an acute bullous dermatitis of hands and less commonly of the feet, sometimes caused by allergy towards epidermophytes or pyogenic cocci or by endocrine, metabolic or sympathetic dysfunction.

Plates 8 and 9 illustrate a case of occupational dermatitis caused by exposure to ammonia and resembling psoriasis.

Legal Aspects

A diagnosis of ' dermatitis produced by dust or liquids ' can for the purpose of compensation under the Act only be made by a practitioner appointed as examining factory surgeon. The certificate so given holds as a legal document unless challenged by appeal to a medical referee within the statutory period. Industrial disease is for Workmen's Compensation Act purposes regarded as a notional accident, so that subsequent allergy or sensitivity does not affect the issue if the original cause can be associated with occupation (Morgan, 1939).

The wording of the Act is ' dermatitis produced by dust or liquids ' and it is agreed on the medical and legal sides that eczema and dermatitis are one and the same reaction. The Act, however, covers more than these forms of eczematous dermatitis from direct irritation or from sensitization. All forms of eczematous dermatitis, including constitutional and seborrhoeic forms, may be produced or provoked by occupational dust or liquids. Ingram (1939), points out that the legal question is whether a particular workman contracting eczematous dermatitis while working under certain conditions would have suffered from similar trouble had he not been so working. As the law stands at present almost any skin disease may be certified as due to occupation and, although real dermatitis is common enough, certificates have been known to include among other conditions actual cases of eczema, scabies, psoriasis and lichen planus. The medico-legal aspect is difficult and comprehensive reviews with useful practical information are given by Schwartz (1943a), and by Mumford (1934, 1935), who advises that a panel of dermatologists should be available so that a report of a specialized nature on a case under argument could be made. Senior physicians with many years of specialized experience should be appointed to the panel.

In the future there should be closer co-operation between the medical practitioners concerned in the settlement of cases. The Works Medical Officer should have a real knowledge of the actual work performed, the length of exposure and materials used and complete records of previous skin diseases occurring on any process. The Dermatologist can exclude any well-defined constitutional skin condition and in general establish the diagnosis and prescribe treatment. The Family Practitioners should have intimate knowledge of the home circumstances, history of previous skin disease and the development of the eruption. The referee should be an independent dermatologist appointed by the Factory Department.

Prevention

A great deal can be done to prevent occupational dermatitis by precautionary measures applied to personnel and plant.

1. Supervision of personnel.
   Selection by pre-employment examinations.
   Instruction.
   Medical supervision and first aid.
2. Direct protection.
   Factory hygiene and design of plant.
   Protective clothing.
   Barrier creams.
3. Cleansing.
   Provision of adequate washing facilities, with routine supervision.

These measures, which have been reviewed by MacKenna (1936) and Horner (1942), are of the highest importance and require urgent application to industry. A substantial reduction in the total cases of Industrial Dermatitis occurring in the filling factories of the Ministry of Supply was secured by supervision of ablutions, the application of barrier substances, the elimination of susceptible workers, and the development and mechanization of many processes (Rep. Chief Insp. Fact. 1943). Most cases caused by primary irritants can be prevented

FIGS. 8, 9.—Occupational dermatitis produced by ammonia.

by mechanical protection and proper education of workers. The importance of skin cleansers and the abolition of abrasives and hand brushes is stressed in a review of preventive measures by Reuter (1941). Collier (1935) is convinced that the most important factors in the control of occupational dermatitis are the discovery of the susceptibles, the control of excessive heat, and the reduction of exposure to the specific skin irritant. No single method is effective for the purpose and consideration must be given to all types of protection (Bourne, 1944).

Examination of Prospective Employees

This should be conducted by the Industrial Medical Officer who is aware of the particular skin hazards. It is desirable to choose workers with a 'good skin' for occupations known to give rise to dermatitis. A good skin is defined as 'one that is of considerable thickness when raised between the finger and thumb, is not too dry, and has no obvious defect such as pimples" (Factory Dept., 1939). People with established skin disease or a recent history of skin disease especially of the eczematous type or other evidence of susceptibility should be excluded from work with potential skin hazards. Dermatitis is likely to recur (Ministry of Labour and National Service, 1943). It is well known that some people are more susceptible to dermatitis than others. Certain irritants will cause dermatitis in any subject, but the majority require an individual susceptibility (Macleod, 1933). Apart from established skin disease susceptibility to occupational dermatitis may be indicated by either a congenital disability such as ichthyosis or by acquired disabilities as a lowered skin resistance from general illness or morbid cutaneous conditions such as seborrhoea and excessive sweating. Itching caused by any skin disease is likely to predispose to occupational dermatitis. It is generally agreed that the seborrhoeic individual is prone to generalized skin affections and is unduly sensitive to irritants. People with varicose veins are prone to develop a generalized skin eruption. General illness may lower the resistance of the skin; Gardner (1922) has even suggested that general illness proceeded dermatitis in 77·4 per cent. of cases, and Mumford (1936) has said that anaemia and dysfunction of the thyroid predispose to skin disturbances. Pyorrhoea and dental sepsis as well as other foci of infection predispose to and later aggravate eczematous eruptions. Pre-employment examination should take special note of these conditions; and obviously any person who has had dermatitis or gives a family history of dermatitis should not be exposed to any fresh hazards. It is a strange fact, however, that in examining the 2000 people for the chemical industry no family history of dermatitis was ever obtained. Patch tests are of little value in selecting people for employment.

Instruction of Personnel.

A frank statement of the risks involved and the method of contraction of occupational dermatitis should be given to workers in hazardous occupations. They should be instructed in cleanly methods of work; how to control dust and keep their benches clean. Foremen and forewomen should be instructed in the principles of the prevention of occupational dermatitis and in normal skin hygiene. Cautionary notices should be displayed in workrooms and washing rooms (Factory Dept., 1932, 1939).

Inspection of Established Employees

This is a measure of the highest value. Repeated and frequent examination of the skin of all process workers and casual contacts should be instituted to detect early signs of skin lesions. Immediate removal of such cases from contact may prevent the development of extensive disease which results in general sensitivity of the skin, as apart from individual susceptibility, the chance of recurrence varies directly with the severity and duration of the primary or subsequent attacks of dermatitis.

The early reporting of all lesions should be encouraged and propaganda established to promote application for first aid for all cuts and minor injuries.

Factory Hygiene and Design of Plant

This is a problem for the engineer in co-operation with the medical officer and is obviously a first principle. Its aim is to prevent actual contact with material irritants by plant design. The importance of this measure is well illustrated by the fact that 45 per cent. of operatives will contract acne if exposed to the fumes of chlorinated naphthalenes (Jones, 1941). In a small chemical factory employing 80 people, 31 contracted occupational skin diseases during a period of one year due to the small scale of the operations where there was a maximum of skin contact with the materials used. Not enough attention is paid to factory hygiene and plant design at the present time. Mechanization and total enclosure of all industrial processes where known skin irritants are present should receive increasing attention. General measures such as ventilation and temperature control are important. Dust should be curtailed by general or local exhaust ventilation, by wetting and vacuum cleaners. Clean benches, adequate working space and the design of tools with adequate handles to reduce contamination of the hands are necessary matters for consideration. The use of dirty rags for wiping the hands should be forbidden.

Barrier Substances

Barrier substances may be used by themselves or as additional protection with gloves against corrosive substances. They render washing easy so that soap usually suffices for this purpose, but are of doubtful value against continuous and severe exposure and should be designed so as to remain on the skin and provide protection for an adequate period. The problem of the provision of barrier creams is very difficult and they should be selected

for each individual irritant. Their provision without care may be dangerous and a fatty barrier cream may increase the absorption of substances soluble in fat, such as T.N.T., nitrobenzene.

Certain criteria for barrier substances were advanced by the Ministry of Labour and National Service (1943) and preparations should be non-irritating to the skin, insoluble in the substance against which the skin protection is required, easily applied, not slippery and easily removed. Peters (1942) points out that barrier creams may be made attractive by tinting and should be correctly applied in a thick even layer. Mumford (1939), discussing the value of various preparations, advanced criteria for the ideal protective and suggested the use of Halden's emulsifying bases. He advances the following classification of types of skin protective:

(1) Fats and oils—for example, petroleum jelly, lanolin, wax, etc.
(2) Jellies of colloid structure—for example, starch, tragacanth, agar, glycerine, honey.
(3) Emulsions containing mixtures of substances in Groups (1) and (2).
(4) Soap bases—for example, stearates, oleates, and palmitates of alkalis: free alkali is usually present.

Schwartz (1942) has recently reviewed this problem and described the following six classes of protective applications:

1. A simple vanishing cream which, when rubbed into the skin, fills the pores with soap and facilitates the removal of dirt when washing after work.

Stearic acid	20
Sodium carbonate	2
Glycerine	6
Water	78

2. Ointments, emulsions or solutions of 'invisible glove' type, water soluble or water insoluble. Fats or oils may be added to prevent the removal of the water soluble type by perspiration. The water insoluble resins or waxes require a volatile solvent for application and a special cleanser for removal. Possible irritant effect may be minimized by the application of hydrous wool fat cream after work.

Water soluble	
Acacia	5
Tragacanth	5
Borax	2
Water	88
Water insoluble	
Gum benzoin	5
Beeswax	2
Anhydrous wool fat	5
Ethyl alcohol	88

3. Ointments that fill the pores with a harmless fat, and protect against water soluble irritants and petroleum oils and to some extent against greases.

Anhydrous wool fat	70
Castor oil	30
Perfume.	

A wetting agent may be added to facilitate removal and to increase penetration of the cream at the time of application because of reduced surface tension.

4. Protective ointments containing non-irritant chemicals to neutralize irritants:

Boric and benzoic acids to neutralize alkalis.
Soap and magnesium hydroxide to neutralize acids.

5. Protective ointments which permit inert powders to adhere to the skin and form a protective covering.

These are useful against abrasives and allergenic substances, e.g. explosives:

Zinc oxide	5
Talc	5
Iron oxide	1
Irish moss	2
Gum benzoin	2
Water	10
Alcohol	15
Vanishing cream	60

Talc powder may be used to protect the skin against lime (Pether, 1937).

6. Protective ointment against photo sensitizing agents, such as heavy coal tar.

Hydrous wool fat	58
Castor oil	30
Titanium dioxide	5
Methyl salicylate	5
Duponol	2
Perfume.	

Most of the proprietary creams are mixtures of the above cleansers.

Klauder, Gross and Brown (1940) describe a number of protective hand creams and detail the formulae of several which are useful for general purposes:

		Per cent.
I.	Petrolatum	70·0
	Hydrogenated cottonseed oil	30·0
II.	White wax	5·0
	Glyceryl monostearate	12·5
	Hydrous wool fat	5·0
	Sodium silicate commercial solution	5·0
	Ammonium hydroxide, 10 per cent. solution	0·5
	Petrolatum	72·5
III.	To formula II, 5 per cent. by weight of latex is added which produces a rubbery film on the skin.	
IV.	For prolonged contact with water:	
	White wax	10·0
	Hydrous wool fat	5·0
	Sulphonated olive oil	10·0
	Petrolatum	75·0

V. Non-greasy preparations which dry on the skin and do not rub off are indicated for dry work and against dust:

Glyceryl monostearate	12·0
White wax	12·0
Wool fat	6·0
Cholesterol	1·0
Sodium silicate, commercial solution	5·0
Ammonium hydroxide 10 per cent. solution	0·5
Water	63·5

Washing Facilities, Cloakrooms and Clothes

The ideal plan is to provide changing and bathing accommodation at the factory entrance so that workers may enter and immediately change into working clothes. After ceasing work they can bathe and return home in their own clothes. The provision of adequate washing facilities is essential in considering the prevention of occupational dermatitis. Basic information is supplied on the arrangement and equipment of wash-rooms and bathrooms in the Ministry of Labour and National Service Welfare Pamphlet No. 8 (1944). Emphasis is laid upon adequate supervision of the facilities when once provided; this is of importance and often overlooked. Examples of washing facilities in different kinds of factories are given with photographs and information about the provision of soap, towels, nail-brushes and baths. Various types of wash basins and details of drainage are shown. Hot and cold water or mixing valves should be provided. Circular fountains or long porcelain troughs are satisfactory types of wash bowls. A good system is to fit the basins without traps and discharge the waste into a glazed channel which communicates with a trapped gully outside the building. Shallow foot-baths provided with showers in a private cubicle adjoining a dressing-room are excellent examples of bathing accommodation.

Skin Cleansing

The best routine method of cleansing the skin is by the use of a good toilet soap and plenty of warm water. This is used for general purposes at noon and at the end of the day. Schwartz (1942) has recommended a good soap as a superfatted neutral toilet soap containing a wetting agent or synthetic detergent. There should be a minimum of free alkali and the *p*H should be 10 or less in 1 per cent. solution. No silica, quartz, pumice, feldspar, resin fillers or organic solvents should be used. The following formula is given:

Neutral toilet soap	30
Bentonite	30
Santomerse	10
Hydrous wool fat	5
Perfume	1

Klauder, Gross and Brown (1940) stress the adverse effects of the use of various cleansing agents in a review of preventive methods. By the adoption of general preventive measures, and barrier creams it is usually possible to facilitate the removal of general dirt and so prohibit the use of abrasives, solvents, soda ash and bleach and acids which often produce dermatitis by themselves. It must be stressed that if efficient substitutes are not provided workers will, of necessity, use these materials which are generally available in chemical works. Even so it may be necessary to develop and provide special cleansing agents for the particular problems. The removal of dyes is often difficult and, for example, solutions of hypochlorite and bisulphite can be provided for the removal of such staining. Nail-brushes are intended for cleaning nails only and much damage can be done by plying these as scrubbing brushes to the skin. Tar, tar rubber, bitumen, oil, paint, turpentine and grease can all be removed by the use of cotton-seed oil, 50–140 c.c. being placed in a bowl and the hands washed therein. The hands are subsequently washed with soap and water.

A useful description of skin cleansers and measures of prevention and treatment of occupational dermatitis is recorded by Schwartz (1941a). The combination of sulphonated castor oil (*p*H 7·2—oil content 50 per cent.) and a wetting agent such as duponol makes a good skin cleanser and does not defat the skin. The addition of an alkali such as trisodium phosphate 1–2 per cent. increases the cleansing powers and only slightly increases the irritant properties, and a mild perfume such as lavender or lilac makes the mixture more acceptable. The preparation has been used successfully in the same way as liquid soap (Schwartz, 1941b).

The prevention of oil rashes requires special consideration. Ordinary washing with soap and water is not effective in cleansing the skin before and after work and preventing these rashes. The Factory Dept. (1932) recommended that the hands and arms should be washed before work with soap and water and protective clothing carefully adjusted. First aid must be obtained for cuts and sores. After work the hands and forearms should be washed in a lotion having the following composition:

Chlorinated lime (powdered) ..	175 grains
Bicarbonate of soda	350 „
Boric acid	35 „
Water	30 ounces

Before use, mix with ten times the quantity of water. After the use of this lotion the skin should be rinsed well with soap and water.

Mummery (1944) stresses the value of neutral sulphonated castor oil in a 2 per cent. wetting agent as a substitute for the use of soap after work in preventing oil acne. Oil should be protected from dust, strained and renewed when contaminated. It can be treated by filtering and heating to 300° F. Twort (1934) describes the measures which should be taken to prevent mineral oil and tar dermatitis and cancer.

The essentials of satisfactory cloakroom accommodation are detailed and plans illustrating the lay-out and space required are also shown in the

Ministry of Labour and National Service Welfare Pamphlet No. 8 (1944). Means for drying wet clothes are necessary. An excellent type of locker is one with a slanting top preventing the accumulation of litter and with screened doors which facilitate inspection. The importance of supervision and the maintenance of scrupulous cleanliness must be stressed. Dirty and excessive clothes are common predisposing causes of dermatitis, so that regular and adequate laundering should be observed.

Gloves, aprons and trousers can be made of closely woven cotton fabrics. Rubber aprons should be worn for very wet work. Sleeves made of closely woven cotton are useful in prophylaxis and treatment.

Gloves cause skin changes of an eczematous nature by maceration of the skin and should be worn as little as possible and as loose as possible. Care should be taken to see that they are clean and dry and regularly inspected. Talc or other suitable dusting powder can be used inside the glove. Leather gloves render the skin susceptible to fungus infections and a powder should be applied to the hands to prevent this. The following formula is advised by Ferguson (1944):

	gm.
Salicylic acid	5
Menthol	2
Camphor	8
Boric acid	50
Starch	35

Treatment of the Skin after Work

If special agents are used to remove material from the skin, especially if they cause defatting, it is essential to provide a substance to maintain the skin in good condition. For this purpose equal parts of lanolin and olive oil or cotton-seed oil can be used. The application of lanolin to the hands at night is probably one of the simplest and most satisfactory prophylactic measures yet evolved (MacKenna, 1936).

Treatment

It is essential to establish an early correct diagnosis in order that the patient may be removed from contact with the causative irritant. For this purpose a scheme of alternative work should be organized, and it is of benefit in mild cases if the employee can remain at work without reduction of wages. Experience indicates that it is wise to seek the opinion of a dermatologist at an early stage in severe or doubtful cases. The benefits of rest and hospitalization for severe cases, acute or chronic, are striking, and prolonged convalescence will help in preventing recurrence of the disease. MacKenna (1945) has indicated that rehabilitation can be applied to cutaneous diseases.

At the earliest signs of a skin eruption the patient's skin should be adequately cleansed and all his clothes changed. The irritation produced by the eruption causes scratching which leads to aggravation, secondary infection and loss of rest. The advantages of treatment by sedatives such as bromides and luminal are well known, and phenacetin and phenazone are useful. If the disease is very acute a single dose of 15 grains of chloral hydrate may be effective. It is doubtful if thiosulphates are effective as non-specific desensitizers. Generally adequate meals with additional vitamin C are required and special diets appear to have little practical value unless the dermatitis is associated with some general disease such as diabetes or nephritis. Hellier (1938) and Wallace (1938) discussed the value of diet and internal treatment in skin diseases. They state that in eczema there is possibly a tendency to acidosis. Excess sodium chloride favours eczema, the sodium ion being particularly important, while calcium, magnesium. and possibly potassium have the opposite effect. The disease is accompanied by a retention of fluid in the skin and this is promoted by carbohydrates and fats and opposed by proteins, and is also affected by the total intake of the patient. The administration of alkalis and the avoidance of sodium chloride are recommended and the following mixture advised:

Calcium lactate.
Magnesium chloride.
Potassium chloride aa 10 gr.
Aqua Chlorof. ad ½ oz. t.d.s. p.c.

In eczematous types of lesions the fluid intake and carbohydrate may be restricted. Tea can be taken without sugar, and potatoes, sweets, pastries and bread can be reduced. Plenty of lean meat and milk are advised.

Local Treatment

Certain principles of treatment were reviewed in an excellent article by Haldin-Davis (1935). Many remedies increase such inflammation as is already present. Antiseptics if employed should be highly diluted. The more acute the inflammation the milder the remedy to be used. Protection of the inflamed surface is the first principle. Gentle wiping with cotton wool soaked in liquid paraffin or olive oil should be employed for cleansing purposes as the inflamed skin reacts badly to water. Sulphonated oils are important cleansing agents for workers who have developed dermatitis and the following formula can be employed:

	Per cent.
Sulphonated olive oil	50–60
Light liquid petrolatum	40–50
Gelatin, 25 per cent. aqueous solution	10

In treating septic diseases of the skin it is most important to cleanse the surface from accumulated discharge without wounding the tender epidermis. If the eruption is wet, calamine lotion or boric acid solution may be used; but if the eruption is dry it is better to employ zinc oxide or boric acid ointments. Ung. zinc oxide (B.P.) consists of zinc oxide

with soft paraffin and 15 per cent. of lanolin and is very suitable for all forms of dermatitis. It may be used as follows:

Ung Zinci (B.P.).

Paraff. molle aa 4 drachms.

Lassar's paste is a well-tried and often efficacious preparation. The salicylic acid may be omitted in acute cases and the paste can be softened by the addition of olive oil. Calamine lotion is a popular remedy and may be used for local eruptions or widely employed on gauze. The sulphonamides are effective in certain diseases of the skin, but it is questionable if their use as local applications in superficial infective dermatoses possesses any advantage over rational treatment with older and safer remedies (Barber, 1944). Taylor and Hughes (1944) described the treatment of infective dermatoses with penicillin applied locally in adequate dosage. It is effective in certain skin diseases caused by cocci. Starch poultices are effective in the treatment of acutely inflamed and crusted areas. In cases where old patches are present 1–2 per cent. of liquor picis carbonis in the ointment may produce improvement. Occasional painting with 1–3 per cent. silver nitrate in water will cure troublesome fissures and cracks.

X-ray therapy is a most valuable agent especially in the treatment of chronic dermatitis. The dosage in acute cases is usually 50 r., repeated every second day and for less acute disease 60–80 r. twice a week. A total of 400 r. should not be exceeded during any period of one month followed by a rest of at least four weeks.

Summary

A review is given of 92 cases of occupational dermatitis occurring in a period of 10 years in a chemical works employing 2000 people.

Sixty-seven cases were produced by primary skin irritants and 25 by secondary skin irritants.

The diagnosis of occupational dermatitis may be difficult and depends on a full consideration of the mode of development of the eruption, its distribution in relation to areas exposed to irritation, the exact nature of the work and irritants handled, and the materials used to cleanse the hands, together with the patient's previous skin history. It is impossible to establish a diagnosis from the appearance of the eruption in a high proportion of cases. Certain criteria should be satisfied; the causative agent identified if possible and classified skin diseases must be excluded. Patch tests are of corroborative but rarely of primary diagnostic value. A list is given of the chief skin diseases to be considered in differential diagnosis. An account is given of the legal situation and suggestions made for closer co-operation between the medical practitioners concerned in the settlement of cases. Methods of preventing the disease are discussed. No single method is effective, and consideration must be given to all types of protection. Examination of prospective employees, instruction and inspection of employees, attention to factory hygiene and design of plant and the provision of barrier substances are important measures. Adequate washing facilities, cloakrooms and clothes should be provided. Adverse effects frequently result from the use of improper cleansing agents and consideration is given to the provision of efficient materials.

The principles of treatment are reviewed and the value of the opinion of an experienced dermatologist is stressed. The benefits of rest and hospitalization for severe cases are striking.

The literature is reviewed.

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BOOK REVIEWS

CANCER OF THE SCROTUM IN RELATION TO OCCUPATION

By S. A. Henry, M.A., M.D., F.R.C.P., D.P.H.

(Oxford University Press, London. 1946. Pp. 112. Price 15*s*.)

A Doctor of Medicine practising in the Manchester area, who has had textile workers as patients, cannot have failed to notice the abnormally high incidence of cancer of the scrotum. The author has been a General Practitioner, an Examining Surgeon in a Lancashire town, and later a Medical Inspector of Factories, so he has had what must be unique inspiration and opportunity to write this monograph which is a detailed analysis of 1631 fatal cases of cancer of the scrotum occurring among the total population of England and Wales between 1911 and 1938.

First reference to the disease was made in 1775 by Percival Pott, who remarked that this was a disease to which chimney sweepers were peculiarly liable. His observations were confirmed by later writers.

Others have described cases in other trades, such as tar distillers and shale oil workers. In 1907 the disease received statutory recognition, and the definition ' scrotal epithelioma occurring in chimney sweeps and epitheliomatous cancer or ulceration of the skin occurring in the handling or use of pitch, tar, or tarry compounds ' was added to the Third Schedule of the Workmen's Compensation Act.

In 1925 a Departmental Committee of the Home Office concluded that epitheliomatous ulceration among mule spinners was due to mineral oil thrown off as a spray from the spindles. Epitheliomatous ulceration was made notifiable in 1920, and since then 3333 cases were notified up to the end of 1943. Of these 40·6 per cent. were scrotal; pitch, tar, and bitumen were thought to be causal agents in 56·7 per cent. of all cases, and mineral oil in the remainder.

Cancer of the scrotum contributes less than 8 per cent. of all deaths caused annually by cancer of the skin and 0·28 per cent. of all deaths caused by cancer of all sites. The official figures given by the Registrar-General for the number of deaths attributable to cancer of the scrotum in England and Wales for the period under review is 1638. Further inquiry of cases of cancer of nearby or ill-defined sites showed that more cases could be added to the Registrar-General's list, but that 85 had to be excluded. Both sets of figures are given in graphs, which suggest that the peak period is about the year 1927. As can be expected, the highest incidence of 754·7 per million occurs among chimney sweepers, and other high incidences occur among patent fuel workers (504·2), cotton mule spinners (422·5), all textile spinners and piecers (315·0), tar distillery workers (212·9), and sack makers (110·2). All in these trades come into contact with tar, pitch or mineral oil. Groups, other than those coming into contact with tar, pitch or mineral oil, showing an incidence well above that for the general population which is 4·2 per million, are makers of glass and sheep dip. These latter groups come into contact with heat radiations and arsenic.

Some indication as to the time necessary for the production of primary growth is given in a study of approximately 1200 cotton mule spinners who developed the disease. The largest number of cases occurred some 49–50 years after commencing work. The minimum time from starting work to the manifestation of the disease was 16 years, and the maximum period recorded was 75 years.

The best method of prevention is to replace cancer-producing products by much less carcinogenic or non-carcinogenic ones. Other methods of prevention include mechanical suppression of dust or fume, either by enclosure of machines or by locally applied exhaust ventilation and cleanliness. Whatever measures may be taken now it is necessary to ensure early treatment of those who have been exposed to tar, pitch or mineral oil in the past. It is a heart-rending experience in Lancashire to find a mule spinner consulting a doctor for the first time with an inoperable growth. This can be prevented by periodical medical examination of workers engaged on processes in which it is known that the disease is likely to occur. Thus the disease may be detected in its earliest stage and successfully treated. This is easier said than done, for many workmen object to these examinations because of suspicion and ignorance. But it is heartening to read a recent report * of the Joint Advisory Committee of the Cotton Industry, on which workers, employers, and the Factory Department are represented. One of their sub-committees recommend that there should be periodical medical examinations of all persons engaged on mule spinning.

The author may have had unique opportunity and inspiration to do this work, but anyone who reads it can well understand that it was no light task ferreting out the egg dealer and the musician who proved to be mule spinners. It is a valuable study to which much time and trouble has been given, both in the collection of facts and the choice of photographs illustrating various types of growth and the trades in which cancer of the scrotum predominantly occurs. The reproduction of an old print of Seyley, the chimney sweeper, and his boy will remind readers that dirty trades and child labour existed long before the industrial revolution.

R. S. F. S.

THE RHEUMATIC DISEASES

By C. D. Kersley, M.D., F.R.C.P.

(William Heinemann, London. 1945. Second Ed. Pp. 155. 15*s*.)

This book is intended for the general practitioner and senior student. It is a second edition but has been so extensively rewritten that it is virtually a new book. Such a book is greatly to be welcomed, and as industrial medical officers know, the rheumatic diseases are among the most important causes of ill-health and loss of time from work. The author in his introduction states that one-sixth of the invalidity of the insured population is due to rheumatic disease, which is responsible for the loss of 3,000,000 week's work and some £2,000,000 to the country each year. Chapters are divided according to the various diseases; this is good. The description of these diseases is also good, though it is surprising that no reference or account is given of Walter Bauer's classic work on changes in the knee joint at various ages. Much attention is given to the place of physical medicine in treatment but, valuable as this is, it must be remembered that it is purely a palliative form of treatment and only tends to underline our ignorance of the causation of this group of diseases. A good deal of attention is paid to rehabilitation and it cannot be over-emphasized that treatment of any disease is not completed until the patient is again capable of earning his living. This little book achieves its object admirably, and is greatly to be recommended to those whom it sets out to serve.

K. M. A. P.

* Ministry of Labour and National Service (1945). Interim Report of the Joint Advisory Committee of the Cotton Industry (Mule Spinners' Cancer and Automatic Wiping Down Motions).

BRITISH AND FOREIGN OFFICIAL PUBLICATIONS

Tetryl Dermatitis. MURRAY, H. M. L., PRUNSTER, R. W., and Anderson, R. D. (1944). Department of Labour and National Service, Commonwealth of Australia, Industrial Welfare Division. Technical Report No. 2.

Data are given resulting from the survey of C.E. rash in two Australian fuse-filling factories. The highest risks of dermatitis are in occupations where tetryl comes in contact with the skin: airborne tetryl plays a very small part in causation. The natural reflex action of touching the face is not readily overcome, and it provides a real source of tetryl contact with the areas of skin usually affected by the rash. The concentration in the atmosphere of jobs such as stemming and pelleting, which have the highest risk as well as the closest contact, was in the region of 0·62 mg. per cu. ft. The major risk of contracting the rash occurred between the fourteenth and twenty-fourth day of service; the greatest risk was in the spring months, while the smallest was in midsummer. Reduced care in personal hygiene during the colder months, and a higher sweat-flow in summer flushing the tetryl from the skin, may be the explanation of this incidence. Tetryl has no effect on menstrual mechanization, and the enforcement of strict observance of the approved routines for washing before meals and bathing before leaving work are the best prophylactics.

Toxicity and Potential Dangers of Aerosols, Mists, and Dusting Powders containing DDT. Various Authors. U.S. Publ. Hlth. Rep., Supp. No. 177.

Acute toxicity of DDT aerosols was assessed by subjecting guinea-pigs, rats and mice to a concentration in air of 12·44 mg. per litre for 45 minutes; only the mice were affected, but they had licked their contaminated fur. The toxicity for mice is increased by increasing the percentage of fatty oil in the aerosol mixture. They died with increased excitability, tremors and clonic-tonic convulsions after a latent period, and the 90 per cent. of Freon in the aerosol was excluded as the cause of these signs. Dogs, rats and guinea-pigs will withstand a concentration of up to 54·4 mg. per litre of DDT, but if 10 per cent. cyclohexanone is used in the aerosol it causes narcosis. Dogs repeatedly exposed to 12·2 mg. per litre of air survived, but mice in the same conditions died unless contamination of their fur was prevented; they showed histological changes in the liver, kidneys, spleen and anterior horn cells. Repeated intermittent exposure of monkeys to 0·183 mg. per litre caused no toxic manifestations, though again it affects mice. Human subjects are unaffected by exposure in a sealed chamber to repeated doses of 10·4 g. of a 5 per cent. DDT aerosol in 10 per cent. cyclohexanone and 85 per cent. Freon, when this is applied for five consecutive days; but the sustained concentration of DDT only reaches about 6 per cent. of that calculated, as most of it settles out of the air within 5 minutes.

Toxicity of DDT dusts was tested by exposure in a chamber or by nasal insufflation. By the former method 10 per cent. DDT dust at 12·13 mg. per 100 litres of air was harmless for dogs even when repeatedly used. By insufflation into the nostrils in daily doses of 100 mg. of pure DDT per kilo over a period of 7 weeks, or by feeding the same quantity by mouth, it was found that dogs were little affected, but if larger doses are given by insufflation, signs of poisoning appear and are preceded by laboratory signs of renal and hepatic insufficiency. Rabbits were then exposed to a heavy mist of 1 per cent. DDT in Deobase for 48 minutes daily over a period of 4 weeks, but they suffered no ill effects beyond irritation of the mucosae and skin caused by the solvent, which is a petroleum derivative.

The solubility of DDT in water was about 1 p.p.m. A volumetric and another chemical method are described for its estimation. Methods for sampling DDT in atmospheres and an alternative method for its estimation by spectrophotometry are both outlined; its x-ray diffraction pattern is shown and described. Apparently 1–5 per cent. of DDT in an aerosol used as an insecticide should offer no serious health hazard, though it is toxic in solution in fatty oils. It should be harmless as a 10 per cent. dusting powder against lice, or as an intermittent mist spray against flies.

The Biological, Hygienic and Medical Properties of Zinc and Zinc Compounds. HEGSTED, D. M., MCKIBBIN, J. M., and DRINKER, C. K. (1944). *U.S. Publ. Hlth. Rep., Supp. No.* 179.

This is a full review of the effect of zinc: (*a*) the role of zinc in biology; (*b*) the zinc content of foods and tissues; (*c*) the effect of zinc on animals other than man, and upon plants and micro-organisms; (*d*) the contamination of foods and water supplies by zinc; and (*e*) the toxicity of zinc to man. With regard to the last heading, there are no new facts about metal fume fever or zinc chills. This annoyance is readily prevented by adequate ventilation, and it is estimated that the chills will not occur if the amount of zinc in the atmosphere is not allowed to get above 15μg./cu. metre of air. There is no good evidence of lung damage from zinc. Zinc chromate produces a very troublesome dermatitis, and zinc chloride and sulphate, being caustic, produce skin irritation. There is some evidence that the continuous ingestion of soluble zinc compounds, swallowed with the saliva, produce a chronic gastritis with vomiting, which does not improve until the man is given work in an atmosphere which does not contain zinc.

ABSTRACTS

Silicia Hazard in Arc Furnace Construction. FIRMAN, E. M, (1946). *Industr. Med.*, **15**, 14.

Ninety-five per cent. of the risk of silicia is eliminated by using prefabricated silicia blocks instead of silica brick chipped to the proper size. Greater durability and less time, used in construction, results.

Irritating Vapours produced by burning Cellulosic Materials. EASTON, W. H. (1945). *J. industr. Hyg.*, **27**, 211.

Persons harmed by inhaling the atmosphere of an enclosure in which substances rich in cellulose such as wood, cotton and paper have been burned, usually suffer from the effects of heat, of carbon monoxide, or a deficiency of oxygen. Under certain conditions, however, the atmosphere may contain a high percentage of organic irritants such as acetic acid, formaldehyde, methyl alcohol and furfural. When the smoke is very hot it will destroy tissues by burning, regardless of its composition, but when somewhat cooler the irritants present attack the mucous membrane of the respiratory tract and create conditions favouring the onset of pneumonia. Illustrative cases are cited in which the smoke inhaled caused serious respiratory injuries without thermal burns, e.g. in some of the 492 persons killed in 1942 in the Coconut Grove tragedy, Boston.

Sampling and Determination of Methanol in Air. ROGERS, G. W. (1945). *J. indust. Hyg.*, **27**, 224.

Solvents containing methanol (methyl alcohol) are used extensively in industry, and the determination of the methanol in the atmosphere is frequently necessary. The presence of 200 p.p.m. has become widely accepted as the maximum permissible concentration for continuous exposure during an 8-hour day. The method of estimation of methanol used by many investigators is to oxidize it with potassium permanganate, followed by the production of a coloration with Schiff's reagent (a solution of basic fuchsin containing sodium bisulphite and sulphuric acid). The efficiency of the various methods used for collecting the samples of contaminated air has not been estimated, and the object of the present investigation was to determine this efficiency, together with the optimum conditions of certain stages of the analysis. A spectrophotometer was used as the colorimeter, the wave-length chosen being 580 millimicrons. A period of 3½ hours was estimated to be the best for the colour-development time, and 1 hour for the oxidation time. The sampling devices studied were (*a*) the midget impinger, (*b*) a fritted glass bubbler, and (*c*) a tower packed with wet glass beads. The dimensions of this apparatus are described in detail. An atmosphere of known methanol content was generated in a continuous flow apparatus, and passed through two absorbers in series. The methanol trapped in the first absorber, with a sampling rate of 1–2 litres per minute and atmospheric concentrations of 200 p.p.m. and 400 p.p.m., was 92 per cent. of the total amount absorbed. The fritted glass bubbler showed an efficiency of 91 per cent. at 200 p.p.m., and 96 per cent. at 400 p.p.m. Efficiencies of 37–87 per cent. were obtained with packed towers, and the performance was variable.

La Maladie des Rotativeurs. DROUET, P. L., and LONYOT, P. (1945). *Arch. Mal. profess.*, **6**, 127.

An account of 'dead fingers' occurring in a shoemaking factory among workers using a vibrating machine known as a 'rotative' is given. The frequency of the vibrations transmitted was in the neighbourhood of 15,000 per minute. During the first days at work on the machine there is tingling of the fingers which persists, and this is followed by whiteness, coldness and loss of sensation. This is worse in the morning, and lasts for a period of 2 hours. After an hour at work the symptoms may disappear. One man who stopped working with the machine in 1934 still develops the symptoms in cold weather or if he puts his hands into cold water. Treatment is ineffective; vascular dilators and sympathetic sedatives are without effect; only radiotherapy to the cervical sympathetic centres produces a slight relief of symptoms.

A Case of Mustard Gas Keratitis treated with Curettage of the Cornea for the removal of a band-shaped crystal-line Deposit. NEAME, H. (1945). *Brit. J. Opthal.*, **29**, 102.

Owing to the corneae of severe cases of mustard gas keratitis being liable to recurring ulceration, they are usually regarded as inoperable. An account is given of a man aged 56 who had a band-shaped crystalline deposit removed from the left eye by curettage. Vision improved from R.V. 6/24, L.V. 3/60, to R.V. 6/18, L.V. 6/36.

Diagnosis of Industrial Poisoning. HAMILTON, A. (1945). *Calif. West. Med.*, **62**, 110.

Alice Hamilton stresses the need of a full occupational history and also points out that toxic substances that are manufactured can be used in the home under trade names. Diagnosis of the unusual is the most difficult. Besides blood changes, dial painters may get osteosarcomata after a considerable interval. Benzene can give rise to all types of blood change. Carbon monoxide anoxaemia may result in death of those cells which cannot stand the lack of oxygen. Most cells when destroyed can be regenerated, but not those of the grey matter of the central nervous system. Vessel walls may be weakened. Atrophy of the optic nerve has been observed after gassing, and cases of cerebral haemorrhage often follow gassing in a few months. Forms of lead poisoning in which the joints and not the muscles are involved are obscure. In human beings, the first symptoms of carbon tetrachloride poisoning can be attributed to the kidneys, and death in uraemia is common. Lead is a germ-cell poison, and Japanese workers have shown that paternal lead poisoning can be conveyed to the offspring. Lead has been found in the blood of the foetus. There is a greater degree of sterility in the wives of lead workers than in the wives of non-lead workers.

Acute Phosphorus Poisoning: Report of Case with Recovery. CHRETIEN, T. E. (1945). *New Engl. J. Med.*, **232**, 247.

A young woman aged 23 swallowed rat paste which contained 130 mg. of phosphorus. She developed nausea, headache, severe abdominal pain and vomiting, and later became jaundiced. There was abdominal tenderness, but the liver was not palpable. B.P. 110/70. Urine: S.G. 1022, no albumin, but reduced Benedict's. Sedimentation rate, 4. Red cells 4,200,000, haemoglobin 86 per cent., white cells 1200 (68 per cent. lymphocytes, 24 per cent. polymorphs). Non-protein nitrogen, 47·7 mg. per 100 ml.; icteric index, 37; bilirubin, 9 mg.

per 100 ml. Prothrombin time, 20 seconds. Bromsulphthalein test normal. She was treated with high-carbohydrate low-fat diet. Daily intravenous infusions of 10 per cent. glucose in saline, preceded by 20 units of regular insulin, were given. Calcium gluconate 1 gm. was given orally three times a day. The patient also received pentnucleotide, ascorbic acid and thiamine. She recovered completely.

The Toxicity of 2,2-*bis* (*p*-chlorphenyl) 1,1,1-Trichlorethane (DDT). CAMERON, G. R., and BURGESS, F. (1945). *Brit. med. J.*, **1**, 865.

In animals a large single dose of DDT produces signs of intoxication in 12–24 hours. The animal is cold, its fur ruffled, and diarrhoea may be present. It is nervous and sensitive to stimuli. Muscular weakness and coarse tremors develop. Movement becomes restricted, staggering, and often spastic, though the fore limbs are rarely affected. Anorexia leads to rapid loss of body weight. Death may occur within 48 hours. With repeated administration of DDT there is loss of body weight, anorexia and diarrhoea: tremors and weakness come later with big doses. The liver is seriously damaged: there are numerous areas of focal necrosis, or large areas of centrolobular necrosis are uniformly distributed throughout the organ. Leucocytosis is an intimation that the toxic level is being reached. In rabbits exposed to large skin doses of DDT evidence has been obtained of a rise in blood calcium values. DDT is tolerated in fairly large amounts, and toxic levels are not easily reached when dilute solutions suitable for insecticidal purposes are employed. Trials on 52 soldiers wearing undergarments impregnated with 1 per cent. DDT suggest that there is little risk of skin irritation and general systemic effects attributable to DDT even with prolonged exposure in ideal conditions for absorption from hot sweaty skin. Danger to health is likely to arise only from careless use of concentrates.

Acute Poisoning due to Petrol Vapour. LAWRENCE, J. S. (1945). *Brit. med. J.*, **1**, 871.

In 1942 a boring was made alongside some petrol tanks from which there was a leak. A man at the bottom of the boring was reeling drunkenly, and when his mate reached him he was unconscious. His mate managed to get him out but, being dazed, he fell in himself from the top. He managed to reach the top again, but lost consciousness and was pulled out by his mates. Both recovered and were well 2 years later. Mild poisoning from petrol causes excitement, euphoria and fits of laughing or crying. Men sometimes become irritable and prone to quarrel. Higher concentrations produce irritation of respiratory mucosa, cough and tickling in the throat, and later ataxia, drowsiness, headache, vertigo, nausea, weakness and irritation of the skin. Vision is blurred, and burning pain is felt in chest and abdomen. There may be tonic contraction of the flexors of the hands and twitching of the muscles of trunk and limbs. With large concentrations hallucinations, delirium and coma develop. Death is from respiratory failure. Mild neurasthenic symptoms, such as headache, sleeplessness and anorexia, are common sequelae of petrol poisoning. Evidence of organic change in the nervous system is not infrequent, especially epilepsy; also lesions of the pyramidal, cerebellar and sensory tract, simulating disseminated sclerosis.

Positive Pressure Respiration in the Treatment of Irritant Pulmonary Oedema due to Chlorine Gas Poisoning. HARDY, G. C., and BARACH, A. L. (1945). *J. Amer. med. Ass.*, **128**, 359.

A boy aged 11 years inhaled a single inspiration of freshly prepared chlorine. Painful persistent coughing ensued for 2 hours; this was followed by laboured rapid breathing, exhaustion, vomiting, and rapid thin pulse. Five hours after the accident he was put in a tent with an atmosphere of 40 per cent. oxygen. Fifteen hours later he was bubbly, with a respiration rate of 44 and cyanosed. The meter mask was applied with 4 ml. of water pressure and 50 per cent. oxygen. Half an hour after positive pressure was applied the cyanosis and headache disappeared and the boy was cheerful and comfortable. The favourable clinical response demonstrated that positive-pressure respiration was the specific therapeutic agent in the recovery of this patient.

Dermatitis from Cutting Oils. WHITWELL, G. P. B. (1945). *Lancet*, **1**, 529.

Some oils produce oil acne, others do not. The author considers that it is due to chemical irritation. Non-soluble cutting oils thinned with paraffin produce severe oil acne and severer eczematoid rashes. It seems possible that the follicle-irritating (acne-producing) and eczematizing tendencies of oil run parallel.

Toxicology of Dichloroethane, HEPPEL, L. A., NEAL, P. A., ENDICOTT, K. M., and PORTERFIELD, V. T. (1944). *Arch. Ophthal.*, **32**, 391.

Guinea-pigs, mice, rats and rabbits were killed by a few exposures of 7 hours to a vapour of 1,2-dichloroethane as low as 400 parts per million. Dogs remained in good condition after nearly 200 exposures at that concentration, but some animals died after exposure to 1000 parts per million. A single exposure of 7 hours to 1000 parts per million led to symmetrical turbidity of the cornea in 8 out of 10 dogs. The clearing process sometimes took 3 weeks for partial regression. The clearing was from the periphery inwards. The development of the turbidity was always equal on the two sides, but the clearing process was sometimes limited to one cornea. No turbidity occurred in cats, monkeys, chickens or in other rodents. When the dogs were given repeated daily exposures of 1000 parts per million in series of 5 exposures separated by rest periods of 2 days, they gradually became tolerant of the vapour. Eventually no cloudiness developed after the exposures.

Acute Fluoride Poisoning, RABINOWITCH, I. M. (1945). *Canad. med. Ass. J.*, **52**, 345.

A man aged 39 attempted suicide by ingestion of sodium fluoride. He had muscle spasms in the feet and hands similar to those occurring in tetany. His stomach was washed out and 15 gm. of calcium lactate was left in. He lost consciousness and died of respiratory failure 3 hours after taking the poison. A few minutes before death his blood calcium was 2·6 mg./100 ml. Sodium fluoride is a corrosive and general protoplasmic poison, and combines with calcium in the ionic state to form insoluble calcium fluoride: it thus lowers blood calcium and produces low calcium tetany. It is also anticoagulant, and affects thrombin formation and injures the liver. It is only slowly excreted, and tends to be deposited in bone, making it white, hard and brittle.

Toxicity of Cresylic Acid-containing Solvent. KLINGER, M. E., and NORTON, J. F. (1945). *Nav. med. Bull., Wash.*, **44**, 438.

Cresylic acid is a toxic substance which may be absorbed through the skin. A man aged 41 cleaned some torpedo gear trains for 6 hours with a solvent containing cresylic acid. His hands were unprotected. He developed dryness and stiffness of the skin of both hands; also watering of the eyes. Later the skin became yellow and cracked, and then peeled. He also had difficulty in expectorating, and there was drooping of the right lower eyelid and retraction of the left corner of the mouth. Finally he developed a right facial paralysis with ptosis.

Nitrobenzene Poisoning, CHAMBERS, J. V., and O'NEILL, E. J. (1945). *Nav. med. Bull., Wash.*, **44,** 1112.

Nitrobenzene is readily absorbed through the skin and mucous membrane of the intestinal tract. An account is given of a fatal case of poisoning through the ingestion of commercial spot-remover containing nitrobenzene in a male aged 20. He had respirations of 10, a thready pulse of 110 and blood pressure 112/40. There was deep cyanosis of the entire body, with twitchings of both arms and muscles of the face probably due to cerebral anoxia. He was treated with gastric lavage, nikethamide and blood transfusion. Necropsy was distinguished by post-mortem lividity of a dark purplish colour and marked congestion. The kidneys were of a chocolate-brown colour. Chemical examination of the stomach contents, liver, brain, urine and kidneys showed the presence of nitrobenzene. The patient died with respiratory paralysis.

Heat Disease, BORDEN, D. L., WADDILL, J. F., and GRIER, G. S. (1945). *J. Amer. med. Ass.*, **128,** 1200.

In a hot humid atmosphere with low wind velocity the incidence of heat-stroke is high. The initial effects are mild but often insidious. First come heat cramps, then heat prostration, with weakness, slight fever, anorexia, headache, abdominal distress, nausea, vomiting and visual disturbances; heat exhaustion, with circulatory failure; and heat-stroke, representing a failure of the heat-regulating mechanisms. The last stages may be abrupt and carry a high fatality rate. When the temperature rises above 86° F. vaporization of water becomes increasingly the predominant means of heat loss, until at 95° F. it is practically the sole means. The wet-bulb temperature seems a more sensitive indication of the probable incidence of heat disease than the relative humidity *per se*, and physical activity should be on a reduced scale at a wet-bulb temperature of 75° F. and should stop at 79°. In the treatment of severe cases intravenous administration of plasma is advised rather than saline, owing to its ready diffusibility in the tissues. One hundred per cent. oxygen by mask combats both the reduced oxygen-carrying capacity of the haemoglobin and the tissue-oxygen defioit.

Is Mononucleosis a form of Myelotoxicosis caused by Benzene? MAZEL, P., PICARD, D., and BOURRET, J. (1944). *Arch. Mal. profess.*, **6,** 18.

A report of the case of a youth of 19 who, until the last year, had been a shoe-maker. For 1 year he had worked with a benzene-containing paste and had complained of increasing lassitude and shortness of breath on exertion. Blood counts showed a marked leucocytosis, at first entirely due to lymphocytes, but later giving a picture similar to monocytic angina. There was a mild anaemia with some normoblasts. The syndrome disappeared some weeks after removal from contact with benzene.

'Dead Hand' in Users of Vibrating Tools. TELFORD, E. D., MCCANN, M. B., and MACCORMACK, D. H. (1945). *Lancet*, **2,** 359.

Workers using an electrically driven tool which turns at a speed of 2900 r.p.m. developed Raynaud's phenomenon. To this tool are attached either emery polishing-wheels or a burr-shaped metal cutter. The worker steadies the tool with the right hand, but the heavy part of the work in guiding and holding the tool falls on the left hand. Out of 300 workers 75 complained of symptoms and 34 were examined. The attacks were induced by cold: there was sudden blanching and deadness of one or more fingers, though the thumb was never involved. After a variable interval the whole gives way to a deep leaden cyanosis. One patient was subjected to sympathectomy, but the result was not good. An attempt has recently been made to damp out the vibrations by sheathing the tool in a sleeve of gas-mask tubing, but in view of the long incubation it is suggested that workers should not be employed in this type of work for more than 9 months. Degenerative changes in the vessel wall or derangement in the function of the arteriovenous anastomoses are suggested as possible causes.

Polycythaemia produced by Cobalt in the Duck. DAVIS, J. E., MCCULLOUGH, A. W., and RIGDEN, R. H. (1945). *J. Lab. and clin. Med.*, **30,** 327.

Young ducks weighing between 150 and 425 gm. were treated with a 2 per cent. aqueous solution of cobaltous chloride given subcutaneously. One group was given 1 mg. of cobalt per 100 gm. of body weight each day for 14 days, and a second group was given 4 mg. for 28 days. Polycythaemia accompanied by early marrow hyperplasia and an increase in extramedullary erythropoietic masses in spleen, liver, kidney and suprarenals resulted; though with continued injection of cobaltous chloride there is evidence of marrow hypoplasia, disappearance of extramedullary erythropoietic foci, and a lowered phagocytic activity of the reticulo-endothelial elements of the spleen and liver.

Mercury Poisoning in Tungsten-molybdenum Rod and Wire Manufacturing Industry. LEWIS, L. (1945). *J. Amer. med. Ass.*, **129,** 123.

A specialized manufacturing industry is responsible for the supply of much of the tungsten, molybdenum and tungsten-molybdenum wire and rod used in the electrical and radio industries. The process of heat-treating these metal products involves the use of the metal mercury and introduces the occupational hazard of mercurialism. Three cases occurring in this industry are reported. The patients complained of tremor of the hands, pain and numbness of the upper extremities, painful gums and in one instance gingivitis. One had been employed in contact with the mercury for 8 years, the other two for 5 and 6 months respectively. The urine of one of the men showed a trace of mercury by the dithizone method. Engineering attempts to prevent the disease actually raised its incidence; forced circulation of air through the department increased the vaporization of mercury, subjecting the workmen to even greater exposure than before air-conditioning the room. Prevention and collection of spillage, ventilation to ensure a minimum concentration in the atmosphere, and housing of apparatus within forcibly exhausted hoods, together with supervised personal hygiene, are the most important preventive measures. In treatment, removal from exposure is the first essential. Dental care is important in all showing gingivitis and stomatitis. The prognosis of the nervous symptoms is unfavourable.

Studies on Heavy Metal Poisoning. II. Sodium Citrate Therapy Effective 67 Hours after Uranium Injury. DARNELLY, G. L., ROSS, C. J., MERONEY, W. H., HOLMAN, R. L. (1944). *Proc. Soc. exp. Biol. N.Y.*, **57,** 75.

Twenty dogs were given intravenous injections of uranyl nitrate (0·5 mg./kg.), and following this received daily intravenous injections of trisodium citrate (0·33 ml./kg.) saturated solution—five dogs each for 10 days, five starting simultaneously and five after 17 hours—all survived. Of five starting after 17 hours and continuing for 5 days, four survived; and of five starting after 67 hours, continuing for 5 days, all survived. The average rise in plasma N.P.N. after 7 days was to 180 mg./100 ml., whereas the urinary P.S.P. fell at the same time to 5 per cent. eliminated after 2 hours. The clinical condition of the dogs was good throughout. The kidneys of the dog that died on the twelfth day showed considerable evidence of regeneration. Biopsy revealed that by the third week two-thirds of the damaged tubules were lined by newly formed flattened epithelium, and 9 months after the injection normal broad columnar epithelium lined the proximal convoluted tubules. It is suggested that sodium citrate supplies ions or molecules that keep the vital 'citric acid cycle' of carbohydrate metabolism going while recovery takes place.

Studies on Heavy Metal Poisoning. I. The Use of Natural Radio-activity for Tracer Studies on Uranium. HOLMAN, R. L., and DOUGLAS, W. A. (1944). *Proc. Soc. exp. Biol. N.Y.*, **57**, 72.

The alpha particle activity of standard dilutions of uranium in blood plasma and in unine–blood plasma was determined, and the uranium content of the unknown samples was calculated from these calibration curves. Two adult dogs fed on regular kennel diet received an intravenous injection of uranyl nitrate (5·0 mg./kg.) as well as daily intravenous injections of a saturated solution of sodium citrate (0·33 ml./kg.) for 5 days before and 5 days after the uranium; and two similar dogs were given uranium but no sodium citrate. At the end of 4 hours radio-activity in the blood stream was minimal, and by the end of 24 hours the count had returned to background level in all four dogs. The average amount of uranium passed in the urine in 24 hours was 65 per cent. The two dogs which did not receive citrate died on the 8th and 9th day respectively, while the two dogs which did not receive citrate injections are both alive 12 months after the uranium injections. No satisfactory data were obtained to explain the manner in which sodium citrate protects against a lethal dose of uranium nitrate.

Hygiene of Workshops handling Glass Fibre. CHAMPEIX, J. (1945). *Arch. Mal. profess.*, **6**, 91.

During the last 10 years glass fibre has been used in France as an insulating material because of its heat-resisting properties. It has to a large extent replaced asbestos. Symptoms such as skin irritation and erythema, blepharitis and irritation of the nose and throat have been produced mechanically; but, so far, in a group of workers observed for a period of 6 months no pulmonary changes have occurred. X-ray changes, however, could hardly be expected until many years had elapsed. Catching the glass dust at the level of the weaving loom, good ventilation of the workshop, complete change of clothing before and after work, and the wearing of tightly woven clothing to stop the penetration of the glass fibre are the preventive measures recommended. Shower-baths should be provided so that the workers may wash before going home. Soothing ointments should be applied before and after work to the exposed parts of the body. Eating in the workshop should be prohibited.

What is wrong with Workmen's Compensation. SIMON, R. S. (1945). *Acta med. Orient.*, **4**, 155.

Modern accident insurance should provide centralized treatment for rehabilitation and re-education for industrial injuries. Adequate compensation for accidents should be automatically related to the injured man's earnings ; loss of earning capacity expressed in percentage should constitute the basis of all compensation. Lump-sum payments should be made for minor accidents. Men rendered helpless should have additional compensation. A medical board should be the final authority for the assessment of loss of working capacity; and one expert judge supported by a medical referee should be in charge of the workmen's compensation cases.

Roentgenology of the Massive Conglomerate Lesions of Silicosis. CAMIEL, M. R. (1945). *Amer. Rev. Tuberc.*, **51**, 527.

The author claims that it is possible to recognize a massive shadow caused by silicosis in the absence of the occupational history and typical nodulation. The lesions are subapical or subclavicular, and almost invariably bilateral with a tendency to symmetry. There is a clear zone of emphysema separating them from the hila and chest wall, and there is invariably emphysema in the rest of the lung fields. The position of the lesions is usually longitudinal and may lie at right angles to the ribs. The lesion in the separate lung fields tend to similar density, and often fibrous strands radiate outwards from the lesions. Diaphragmatic deformities are frequent, and limitation of diaphragmatic motion is common. The trachea is usually in the midline.

Methyl Alcohol Poisoning. KAPLAN, A., and LEVREAULT, G. V. (1945). *Nav. med. Bull. Wash.*, **44**, 1107.

Forty-two cases of poisoning are reported. Methyl alcohol was found in Japanese fuel drums and was drunk in fruit juices. Thirteen of the victims died. Most of them consumed between 4 and 8 oz. Nineteen patients had mild symptoms consisting of blurring of vision, moderate headache, nausea, and vomiting. Thirteen patients with moderate symptoms complained of blurring and failing vision, especially for distant objects; abdominal cramps, epigastric distress, vomiting, hiccough, dyspnoea and drowsiness. Memory defects and mental confusion were present in all. Eight with severe symptoms showed coma, cyanosis of the upper portion of the chest, face and finger-tips, cold moist clammy skin, dilated fixed pupils, slow laboured respiration and rapid thready pulse. All died. It is suggested that the toxic effect is produced by oxidation through formaldehyde to formic acid, which has a specific affinity for the central nervous system, particularly the optic nerves and respiratory centre.

Alkali Treatment of Methyl Alcohol Poisoning. CHEW, W. B., BERGER, E. H., BRINES, O. A., and CAPRON, M. J. (1946). *J. Amer. med. Ass.*, **130**, 61.

For 60 years methyl alcohol has been recognized as a poison causing either death or blindness in a large percentage of cases. Two groups of patients, totalling 31, partook of mixtures containing methyl alcohol and showed clinical evidence of poisoning. Five died within 3 hours of admission to hospital, and the rest recovered and were returned to duty. Methyl alcohol is not fully oxidized in the body, but is broken down into formic acid and formaldehyde resulting in a profound acidosis. The diagnosis is usually made on the presence of a toxic state following ingestion of an alcoholic beverage, or other exposure to methyl alcohol, in which impairment of vision exists with dilatation of the pupils. Methyl alcohol and its derivatives can be found in the urine or gastric contents in abnormal amounts. Individual susceptibility to methyl alcohol varies considerably and its action is slow. Post-mortem examination of five fatal cases revealed cerebral oedema, hypostatic pulmonary congestion, fatty infiltration of the liver and passive congestion of all organs.

Excellent results were obtained from treatment of 26 cases. Shock and imminent respiratory failure are treated by the administration of nikethamide intravenously and oxygen. Prompt elimination of the acidosis is the essential part of treatment. Sodium r-lactate molar solution of three chlorides was given intravenously and sodium bicarbonate by mouth. The plasma carbon dioxide combining power determines when the course of alkali will have to be repeated.

It is important to keep the eyes covered until all visual and retinal changes have disappeared.

Clinical Investigations of Methyl Alcohol Poisoning, with Special Reference to the Pathogenesis and Treatment of Amblyopia. RØE, O. (1943). *Acta med. scand.*, **113**, 558.

Work on 16 cases of methyl alcohol poisoning is reported. The action of the poison is attributed to the inhibition of oxidation processes by formic acid, which forms a complex compound with iron. The process is followed by an acidosis which is mainly due to lactic acid. Amblyopia does not occur until the acidosis is well established, except in the presence of strong light. Increase of metabolism may provoke or aggravate amblyopia since it increases the acidosis. The oxidation of methyl alcohol is checked by the consumption of

ethyl alcohol, and all signs of poisoning may be averted if it is drunk before or repeatedly after the consumption of methyl alcohol. Treatment consists, first, in counteracting the acidosis by the intravenous injection of isotonic sodium bicarbonate. Copious fluid by mouth combats dehydration and promotes diuresis, and loss of chloride may be counteracted by 5 per cent. glucose in saline. Gastric lavage should not be undertaken until the intravenous sodium bicarbonate is started. Ethyl alcohol helps to prevent recurrence of acidosis. The eyes require protection against light during the acute stage of the poisoning and if amblyopia persists.

Acute Methyl Alcohol Poisoning. JACOBSON, B. M., RUSSELL, H. K., GRIMM, J. J., and FOX, E. C. (1945). *Nav. med. Bull., Wash.*, **44**, 1099.

A report of 18 cases of poisoning with six deaths from mixing methyl alcohol (intended for use in testing for leaks of Freon gas in refrigerators) with water and fruit juices. The six who died had more than 750 ml. and 12 who lived 90–500 ml. The symptoms were alcoholic intoxication, drowsiness, headache, photophobia, blurring of vision, dyspnoea, nausea, and vomiting within 24 hours. In most patients blurred vision and dyspnoea persisted up to 72 hours. One patient died within 12 hours and five within 48 hours. Ocular abnormalities consisted of various degrees of loss of colour vision initially green colour sense, and loss of central vision. Most urines were acid and contained albumin, acetone, and hyaline and granular casts. The blood contained 1–5 mg. alcohol per 5 ml. Post-mortem findings included cyanosis; intensified post-mortem rigidity; marginal emphysema of lungs; subpleural and subepicardial haemorrhages; small areas of necrosis of liver; oedema and mucosal congestion of stomach and duodenum; marked swelling of kidneys; oedema, swelling, and congestion of brain and pia-arachnoid. The patients were treated with gastric lavage, enemata, magnesium sulphate and repeated intravenous 5 per cent. dextrose in saline and large amounts of sodium bicarbonate, both orally and intravenously. It is suggested that acidosis plays an important part in producing symptoms.

An outbreak of Cadmium Poisoning. JENNER, G. G., and CUNNINGHAM, J. A. K. (1944). *N. Zealand med. J.*, **43**, 282.

An outbreak of poisoning affecting 62 airmen who had drunk a mixture of 6 oz. of dehydrated lemon powder and 46 oz. of canned grapefruit juice in 8 gallons of water and ice is described. The mixture was made in a metal urn with a galvanized interior. Analysis gave Deniges' reaction for cadmium positive, and the Spacu reaction for cadmium estimated by extinction dilution showed 20 mg. per 100 ml.; 2·4 ml. N/10 NaOH was required to neutralize 5 ml. of the mixture; the high acidity of the mixture was responsible for solution of the cadmium from the galvanized vessel. Symptoms developed 15 minutes to 1 hour after taking the drink. They consisted of severe vomiting, acute colicky abdominal pain, and diarrhoea. Syncope occurred, many lying on the ground helpless, pale and sweating. Diarrhoea and vomiting lasted up to 3 hours, and sometimes the chloride depletion caused cramps in the gastrocnemia. Of the 62 men, 42 had fully recovered within 48 hours. The actual amount of metal imbibed by each man was 56 mg. Morphine to combat shock and a generous fluid intake combined with sodium chloride tablets was the successful treatment.

Studies on the Duration of Disabling Sickness. Frequency of Short-term Absences and its Relation to Total Frequency. GALAFER, WM. (1944). U.S. Publ. Hlth. Rep., **59**, 1077.

A statistical inquiry into the frequency of absences lasting less than 4 days and its relation to the total frequency. The absences of male workers in a public utility over a period of 10 years were investigated. The high total frequency which always occurs in the first quarter of each year (52 per cent.) is associated with relatively few absences of less than 4 days; whereas there is a relatively high proportion (62 per cent.) of such absences in the low total frequency of the third quarter. The corresponding figures for the respiratory diseases were 51 per cent. and 64 per cent.; the elimination of the non-respiratory diseases effected relatively little change in the proportions for all sickness. The effect of the winter months on absence frequency is well known; but it is shown that the periodic increase in frequency is not evenly distributed, but is less evident among the short-term absences.

Cases of Exposure to Methyl Bromide Vapours. TOYRANGEAN, F. J., and PLAMONDEN, S. R. (1945). *Canad. J. publ. Hlth.*, **36**, 362.

An account of three cases of poisoning occurring in a plant where fire extinguishers were being filled. The fire extinguishers were either pure methyl bromide or a mixture of 20 per cent. carbon tetrachloride and 80 per cent. methyl bromide. There were two exhaust hoods where the filling was being done. The atmospheric concentrations at breathing level in front of hood No. 1 were 0·79 and 0·63 p.p.m. and 0·45 p.p.m. in the general atmosphere of the room. These were considered to be low; tests were repeated, and concentrations in front of No. 1 hood were found to be 385 p.p.m., 390 p.p.m. and 297 p.p.m., in front of No. 2 19·1 p.p.m., and in the general atmosphere 0·2 p.p.m. and 11·4 p.p.m. The patients had the usual symptoms of methyl bromide poisoning, the blood bromides being 211 mg. and 55 mg. per 100 ml. Improvements in the ventilation, periodical air sampling to verify the efficiency of the new system, and frequent medical examinations of the workers employed in the filling-room prevented further cases.

Drowning in Gasoline. MEAD, S. (1945). *American Journal of Chemical Pathology*, **15**, 342.

The first recorded case of drowning in gasoline with a detailed autopsy report. The worker is presumed to have been overcome by fumes and fallen into the bilge of a tanker. Ten to 16 hours later the skin showed no vesication but the epidermis would desquamate to the touch; this constituted a chemical burn. The lungs were heavy and congested and the larger air passages denuded of epithelium. Little gasoline had been swallowed.

The One-eyed Worker. MINTON, J. (1945). **Brit. J. *Ophthal.*, 29,** 475.

A questionnaire sent to 398 firms employing 1,000,000 workers revealed that one-eyed workers are to be found in all types of industry, even those requiring close work; their efficiency and accident records are satisfactory. Those who lost an eye in childhood are not handicapped even when doing clerical work, precision engineering, or working on scaffolding. Present statutory examination and eyesight tests in juveniles will cause difficulties for the one-eyed in selecting a trade.

A study of those who lose an eye as adults shows that most did so whilst in mining or heavy industries while hammering, chipping, etc. Adjustment to monocular vision, especially depth and distance judging, is quickest in the intelligent and experienced, e.g. engine drivers, and in the young; women are very slow to become adjusted. Loss of the dominant eye, which in 72 per cent. of people is the right, causes the greater disability.

The time needed before returning to work should be 2–3 months. Many men, however, stay away 6–10 months, owing to lack of confidence and fear of losing the other eye, to compensation and litigation difficulties or to the unwillingness of firms to employ the one-eyed. But welfare and safety departments report that, after the initial stages, they are as efficient

as two-eyed people and no more prone to accidents. The under-confident need simple exercises and training. Subsequent choice of employment should depend on the vision of the remaining eye, and the job should not involve a risk to this eye. Close work is possible for those with 6/6 or 6/9 vision, but undesirable at 6/12 or below. It is reasonably safe for the one-eyed to drive vehicles.

Health Services for Hospital Personnel. V. Cost of Illness and Operating Costs of a Personnel Health Service. GEIGER, A. I. (1945). *Yale Journal of Biol. and Medicine*, **18,** 15.

Based on the expenditure in one year (1942–3) of a hospital Personnel Health Service covering 850 employees. Estimated operating costs are under various headings. Firstly, salaries for two nurses, secretary, three part-time medical officers, and fees for consultants totalled approximately $10,000. Secondly, laboratory and x-ray facilities cost $4,103 this included 848 x-ray examinations at $4 each, 714 being of the chest. Thirdly, an estimate of 10 per cent. on the original cost of buildings and equipment is $150, while expendable supplies cost $180 and drugs $284. The total of $13,485 constitutes a cost of $16 per capita, or $2.50 more than for plant health services in industry, though the service is far more comprehensive. Further, the loss to the hospital in maintaining sick employees in wards and elsewhere was $3700, after various contributory schemes had operated. Sickness absence, which implies lost working days and the engaging of outside help, was estimated to cost a further $8000 covering 3500 lost days. The overall cost of the service was therefore about $23,000, or $27 per head, which justifies it economically.

Methyl Bromide Poisoning: A New Industrial Hazard. VINER, N. (1945). *Canad. med. Ass. J.*, **53,** 43.

Methyl bromide boils at 4·5° C. and forms a gas 3·5 times heavier than air. It is effective for extinguishing petroleum and electrical fires, and is used as a refrigerant, as a delousing agent and as an insecticide fumigant. Forty minutes exposure to the vapour in a closed room has caused death. Sometimes 20 per cent. of carbon tetrachloride is added to give a detectable odour, but phosgene may then be evolved if the mixture is used as a fire extinguisher. The literature contains 47 cases of poisoning with 12 deaths. The symptoms of contact with methyl bromide are vesication of the skin, reddening of the eyes, coryzal symptoms and cough following inhalation, and ocular and dyspeptic symptoms with a persistent taste and smell as of burnt rubber. Prolonged exposure leads to weakness and ataxia of the arms and legs, drowsiness and twitching movements, at which stage the danger point has been reached. The next stage comprises fever, drowsiness, convulsion and death in coma. Patients who recover may be mentally slow and subject to muscular twitchings.

Two cases are described. One man was a filler of fire extinguishers who died in coma and who had a high serum bromide level (211·6 mg. per 100 ml.) and whose brain showed multiple haemorrhages; the other survived after exhibiting ataxia, slow cerebration and convulsions, and with a serum bromide of 55 mg. per 100 ml.

Development of a Chemotherapy of Systemic Arsenical Poisoning. DANIELLI, J. F., DANIELLI, M., MITCHELL, P. D., OWEN, L. N., and SHAW, G. (1946). *Nature*, **157,** 217.

In order to find the site of loss of protein from the blood capillaries, serum albumen was tagged with the diazonium hydroxide of a dye of the trypan blue type. About half the loss was shown to occur at the site of the lesion and the rest widely throughout the body, but particularly in the skeletal muscles and viscera. Search was made for a non-toxic drug able to reach the whole vascular system; capable of preventing arsenicals penetrating from the blood stream into cells, and able to remove them if they have penetrated; and capable of being readily excreted when it has reacted with the arsenical. A detoxicated thiol was therefore sought. The detoxication can be achieved by introducing protein groups such as OH or COOH into the molecule. The most satisfactory compound was the O-glucoside of dithioglycerol and was named BAL-intrav. With a lewisite dosage, which would normally kill 95 per cent. of rabbits in between 10–48 hours, BAL-intrav would save 50 per cent. if treatment was delayed 6½ hours, the loss of 50 per cent. being due to tardy removal of arsenic from intracellular sites. However, a dose of 4 mg. per kilo of BAL when given with a high dosage (1–1·5 gm. per kilo) of BAL-intrav reduced the mortality to 25 per cent. A dose of 4 mg. per kilo of BAL produces only transient signs of toxicity and when given without BAL-intrav did not reduce mortality at all. A dose of 100 mg. per kilo given as a single intravenous dose produced no ill-effects in a number of men.

Beryllium Poisoning. VAN ORDSTRAND, H. S., HUGHES, R., DE NARDI, J. M., and CARMODY, M. G. (1945). *J. Amer. med. Ass.*, **129,** 1084.

During the past 4 years 170 cases were seen among workers in three plants. Manifestations included dermatitis, chronic skin ulcer, and inflammatory changes in the respiratory tract. The severest manifestation was diffuse pneumonitis which caused the death of five patients. The incidence and severity of changes were proportional to the degree of exposure and are caused by the chemical irritation of dusts and fumes. Acid radical salts are more toxic than other compounds but severe damage may be caused by beryllium compounds other than the acid radical salts. Rhinitis, nasopharyngitis, contact dermatitis and skin ulcer occurred in 42 patients in contact with beryllium sulphate fluoride or oxyfluoride. The eruption was oedematous and papulovesicular and occurred on the exposed portions of the body. Itching was intense. Inadequate cleanliness, excessive perspiration and failure to use protective devices were contributing causes. After exposure was terminated the condition subsided. Calamine lotion and phenol, wet packs of 10 per cent. Burrow's solution, and boric acid ointment with phenol were the best local treatments. Ulcer resulted from a crystal of beryllium deposited within the skin layers. Treatment was early incision of papule, before necrosis occurred, and curettage of the fibrous base. Healing was complete in 8–10 days. Conjunctivitis occurred as a 'splash burn' or in association with contact dematitis of the face. Local treatment included the use of boric acid solution with mild zinc and adrenalin.

Ninety patients were seen with upper or lower respiratory infections. Soreness of nose and throat and epistaxis were the symptoms of upper respiratory infections. Exposure may be avoided by the use of an air mask. Most cases cleared in 3–6 weeks. Lower respiratory tract infections were characterized by non-productive cough, but with occasional blood-streaked mucoid sputum and dyspnoea. Râles were characteristically present in the early phase of inspiration, fine at first, becoming worse. Vital capacity was reduced as much as 30 per cent. There was low-grade fever. If the patient was removed from contact, no case of pneumonitis developed after 3 weeks of onset. Chemical pneumonitis developed in 38 workers with five deaths. Symptoms were cough with occasional blood-streaked sputum, substernal burning pain, shortness of breath, cyanosis, abnormal taste, anorexia with some weight loss, and increasing fatigue. The onset was insidious. Vital capacity was reduced as low as 2000 c.c. Signs were conspicuously absent, sedimentation rate was normal. Bronchoscopy showed a hyperaemic mucosa with some oedema. X-ray findings showed diffuse haziness of both lungs, followed by development of soft irregular areas of infiltration with prominence of pure bronchial markings, and then by absorption of soft

infiltration and appearance of discreet large or small conglomerate nodules scattered throughout both lung fields, and finally by clearing of the lung fields after 1–4 months. Necropsies showed atypical pneumonitis. Striking features in lung tissue sections were large numbers of plasma cells, relative absence of polymorphonuclear infiltration, diffuse pulmonary oedema and haemorrhagic extravasation; fibroblasts with evidence of organization were present. Beryllium present in the lung varied between 6·20 and 1·89 mg. per 10 gm. of dry tissue. Elimination of fumes and dusts at all stations is necessary to control the disease.

Penicillin in Tetanus. ALTEMEIEA, W. A. (1946). *J. Amer. med. Ass.*, **130,** 67.

The outlook in generalized tetanus is always grave and it is disappointing to see the failure of large doses of antitetanic serum once the disease has developed. The potential value of penicillin in the management of 16 cases of established tetanus was explored. All patients received antitetanic serum intravenously or intramuscularly as soon after diagnosis as possible, and would have received a million or more units of penicillin if they had lived long enough. There were 10 deaths and 6 recoveries, giving a mortality rate of 62·5 per cent. Nine of the 13 patients treated with penicillin died and analysis of the deaths re-emphasize the relationship of high mortality rate and short incubation period. Clinical study of the 16 cases failed to show evidence of any beneficial effect of penicillin on the course of the disease. There was no obvious fall in the temperature or pulse rates, no decrease in mortality or morbidity, nor any definite decrease in the severity, duration and frequency of the convulsive seizures. Improvement may occur if tetanus is complicated by pneumonia or other infections susceptible to penicillin.

The failure of penicillin to alter appreciably the course of established tetanus re-emphasizes several very important concepts: (1) that the length of the incubation period is the most important single prognostic sign; (2) that the essential primary cause of the disease is a bacterial toxin; (3) that successful management depends not on chemotherapy but on early diagnosis during the prodromal period, immediate neutralization by specific serotherapy of all existing free toxin in the body, removal of the source of toxin, control of the convulsive seizures, control of the attacks of respiratory arrest and the administration of general supportive measures.

Occupational Dermatoses: Disability and Compensation. LANE, C. G. (1945). *New Engl. J. Med.*, **233,** 711.

Diseases of the skin comprise 65 per cent. of all occupational disease. There is, however, surprisingly little material indicating the actual amount of disability that occurs in these cases, the expense in terms of compensation paid or the medical costs involved. A large number of workers with occupational dermatitis have no disability, or disability for an extremely short period, but there also occur long-drawn-out disabilities that are extremely costly. Figures are given on duration of disability, wages lost, medical expenses and compensation paid which, though incomplete, are impressive enough to warrant more attention. It is not generally realized in what a high percentage of the cutaneous cases seen by the average physician a causal occupational factor arises, and if all persons, including housewives, were considered, an occupational factor would need to be reviewed in 15–20 per cent. of cases. Criteria for diagnosis are given and difficulties discussed. The type of disability that results from cutaneous disease is varied, and its duration has many contributory factors such as carelessness, late detection and treatment, and additional exposure after the onset of irritation.

Disability can be reduced and many cases prevented and the industrial physician can secure early diagnosis by training nurses and foremen to detect skin manifestations.

A further study should be made to ascertain the actual amount of loss incurred from occupational dermatoses.

Chronic Exudative and Indurative Pneumonia due to Inhalation of Shellac. HIRSCH, E. F., and RUSSELL, H. B. (1945). *Arch. Pathol.*, **39,** 381.

Tradesmen working with sprays of varnish, lacquer and shellac may inhale finely divided particles of these finishing substances. An account is given of a man aged 59 who had been exposed to these and wood dust for many years in a furniture factory. He complained of a chronic non-productive cough, tiredness and weakness for several years, dyspnoea for 9 months, and loss of weight. The necropsy demonstrated chronic diffuse exudate and indurative pneumonia in both lungs, while the characters of the solid lung and a small deposit of fibrin on the visceral pleura suggested a lipoid pneumonia. Ethyl alcohol and ether extraction of the lungs yielded large amounts of a viscid material like shellac dissolved in an oil medium. This material injected into the tracheae of rabbits caused necrosis and marked exudative and fibroplastic inflammation of the lungs.

An Unusual Type of Keratitis associated with Exposure to N-butyl Alcohol (Butanol). COGAN, D. G., and GRANT, W. M. (1945). *Arch. Ophthal.*, **33,** 106.

An outbreak of keratitis occurred among 75 girls in a factory making army raincoats. The operation which caused the trouble consisted in cutting the goods, stitching the buttonholes, and cementing the sleeves, collars and pockets in place. In the cementing process polyvinyl butyral resin paste and butanol were used. The butanol vapour concentration of the air varied from 15 to 100 parts per million. Twenty-eight workers were observed to have corneal changes, and 19 complained of ocular irritation, foreign body sensation, epiphora, and burning of the eyes; the symptoms were worst on awakening in the morning. Blurring of vision, itching, and swelling of the lids also occurred. Both eyes were affected almost symmetrically. The changes were best seen by slit-lamp microscopy, and consisted of clear vacuoles, of diameter 0·05 mm., studded through the most superficial layers of the cornea. There were often as many as 1000. After removal from work for a week the number might be reduced to 100. They recur within a few days after return to work. Exposure of mice, guinea-pigs, rabbits, and dogs to the vapour of butanol or of mixtures of butanol, diacetone alcohol and denatured alcohol was uniformly unsuccessful in reproducing the condition seen in human beings.

About 13 cases of Methyl Bromide Intoxication: 7 with 2 deaths occurred in an Accident. MICHAUX, J., CLERCY, A., and LECHEVALLIER, G. (1945). *Arch. Mal. profess.*, **6,** 141.

Methyl bromide is extensively used as a fire extinguisher and in agriculture, and even in homes as an insecticide. In a factory there have been 13 cases of methyl bromide poisoning, and in one incident 7 cases occurred, one of which died at home and one in hospital. In the premonitory stage dysarthria and painful hypersensitivity to sound are characteristic symptoms, while in the chronic stage ataxia of the upper limbs and myoclonic tremors of the first three fingers of the right hand are characteristic. The usual symptoms, such as vertigo, headache and pseudo-drunkenness also occurred, but it is always several hours after leaving the factory before the symptoms developed. This latent period renders the gas more dangerous, since the worker does not realize what has happened. It is, therefore, all the more important that these workshops should have adequate ventilation. Methyl bromide should not be sold to the general public, but only to those specialized in the use of it. This particularly applies to its use in the disinfestation of houses, where it is important to ensure that pockets of gas do not remain below the windows.

Benzene Poisoning. DINBERG, M. C. (1945). *Canad. med. Ass. J.*, **52,** 176.

A review of the recent literature of the subject, without references, stressing that while short exposure results in hyperplasia, prolonged exposure causes hypoplasia of the bone marrow.

Medical Care of the Industrial Worker. ROBSON, R. B. (1945). *Canad. med. Ass. J.*, **52,** 143.

The purpose is to control plant working conditions, so that an optimum environment may be provided for the worker; an environment in which he can work with safety and under conditions that are not detrimental to his health. Industrial hygiene includes plant sanitation, illumination, ventilation, safety provisions and control of occupational disease. The health of workers must also be taken care of by general medical supervision and by the prevention of physical defects. Finally the worker must be restored to health and earning capacity as promptly as possible following accident or disease.

The Physician's Role in Protecting the Worker's Health through Control of the Industrial Environment. BULMER, F. M. R., and MCCALL, G. R. (1945). *Canad. med. Ass. J.*, **52,** 136.

Physical conditions such as ventilation, heating, lighting, sanitation, seating and noise are first discussed. Most industrial diseases are caused by dusts, gases, vapours and fumes that have poisonous properties. Silica in coal mines and foundries, limestone as an irritant, lead, arsenic and cadmium, are quoted as examples. Poisonous vapours include carbon monoxide, hydrocyanic acid and mercury vapour. It is stated that 1 mg. of mercury breathed daily over a period of time can cause poisoning. Nitrous fumes, benzene, trichlorethylene, carbon tetrachloride, gasolene, hydrogen sulphide and arsine are referred to. It is stressed that records are essential. Attention paid to minor illness and complaints can prevent serious outbreaks of occupational sickness.

Observations on Bromide Intoxication. MILLIKAN, C. H. (1945). *J. Iowa St. med. Soc.*, **35,** 120.

Bromides act by inhibition in cerebral hemispheres. They are excreted by the kidneys, but are also retained in the body and tend to accumulate. An increased amount of bromine in the blood is essential to a diagnosis of bromide intoxication; some authors put the level at 75 mg. per 100 ml., but it probably is in the order of 150–200 mg. The patient complains of a desire to sleep and has difficulty in concentrating his attention; slurring of speech follows, and later a mild staggering gait develops; finally there is clouding of consciousness and stupor, the patient sometimes being disorientated for a time and incontinent. In bromide psychosis, besides the symptoms above, hallucinations, delusions, excitement, negativism and general disorderly conduct occur. The principal treatment of bromide intoxication is to discontinue the drug.

On the Toxicity of Certain Synthetic Rubbers. TRUFFERT, L. (1945). *Arch. Mal. profess.*, **6,** 225.

Organic sulphur compounds give rise to a disagreeable smell and cause irritation of the eyes, and also the upper respiratory tract, and may make work impossible. Tricresyl phosphate is used as a plasticizer in certain varnishes and synthetic rubbers, and if such substances should get into the intestines there is a real risk of toxic manifestations. The author thinks that it is necessary to take all measures to prevent prolonged contact of the hands of workers with tricresyl phosphate.

Sensitization to 'Tetryl.' GELL, P. G. H. (1944). *Brit. J. exp. Path.*, **25,** 174.

Experiments with guinea-pigs show that tetryl sensitizes by reacting with the recipient's body protein to form an antigenic 'picryl protein.' Methods of making 'picryl proteins' *in vitro* are described, and by means of these it was shown that there is no correlation between these and circulating precipitins. Sensitized animals are shown to exhibit anaphylactic phenomena on intravenous injection of picryl proteins both *in vivo* and *in vitro*. Animals were also sensitized by the inhalation of an artificial 'smoke' of tetryl, but guinea-pigs could only be weakly sensitized by the injection intradermally of picryl proteins.

Alterations in the Blood during Chronic Carbon disulphide Poisoning. BINCT, L., and BOULIÈRE, F. (1944). *Arch. Mal. profess.*, **6,** 12.

Apart from the anaemia noted by most investigators, no changes such as low blood calcium or cholesterol, diminished alkali reserve, increase of blood potassium or formation of sulphaemoglobin have been noted by the above authors in poisoning caused by repeated small doses of carbon disulphide. Experiments were therefore carried out on a dog of 8 kg., which was exposed to 1 mg.–1 cg. of carbon disulphide per litre of air daily. These showed that there was always a fall in red cells, but no marked alteration in numbers of white cells or in the differential count. Investigations into the blood sulphur and urinary sulphur revealed little variation. Results showed an increase in the total glutathione in the blood—an increase which persists for several days after the end of the exposure.

The Value of Health Examination in Industry. BRISTOL, L. D. (1945). *J. Amer. med. Ass.*, **128,** 627.

A study of the comparative sickness records of a group of 100 males who, in a period of 14 years, had at least 10 medical examinations, compared with a non-examined control group. No significant difference existed so far as concerned total incidence and specific causes of illness; but there is evidence that the sickness incidence of the examined group improved materially as compared with the non-examined as the years advanced. Thus for major sicknesses 1975 calendar days for the examined group in the first 7 years compared with 1718 in the non-examined, but 2333 compared with 2799 in the second 7 years. Examination also appeared to reduce the duration of illnesses, particularly as the years advanced. Examinations are of great value in finding early cases of pulmonary tuberculosis and diseases such as diabetes, and in correcting physical defects in eyes, tonsils, teeth, and other parts of the body. To get the most good out of examinations persistence is important, since the ultimate values are slow in appearing.

Methionine in Carbon Tetrachloride Poisoning. EDDY, J. H. (1945). *J. Amer. med. Ass.*, **128,** 994.

Eight patients developed carbon tetrachloride poisoning; they were employed at an operation where rags saturated with carbon tetrachloride were wiped over a hot land-mine. The mine was hot enough to vaporize the carbon tetrachloride, but not hot enough to produce phosgene. The concentration in the atmosphere was probably more than 2000 parts per million. Symptoms included a sense of drunkenness, faintness, dizziness, headache, nausea and vomiting, cough and cramping pain in the abdomen, but all the patients had continued work for 6 hours after exposure. One patient died on the fourth day, sections of her liver showing almost total necrosis, a second patient died on the fourteenth day with a fatal pulmonary haemorrhage, and sections of her liver showed diffuse central necrosis that involved one-third to a half or more of the lobule. The six other patients had an icterus index between 2·7 and 344; and

two of them showed the typical picture of liver and kidney involvement. The patients were negroes who lived on a protein-deficient diet. All six patients were given methionine intravenously: the author believes that one of the patients would have died without this treatment, and that the results in this small group of cases are at least suggestive of the clinical value of methionine in certain types of liver damage.

Pathological changes in Animals exposed to a Commercial Chlorinated Diphenyl. MILLER, J. W. (1944). *U.S. Publ. Hlth. Rep.*, **59**, 1085.

Rats were exposed to ingestion, corneal instillations, subcutaneous injections and skin applications of commercial chlorinated diphenyl in doses varying from 17 to 1380 mg., sometimes single and sometimes repeated at regular intervals; guinea-pigs were treated by ingestion and skin applications, and rabbits by skin applications. Fatty degeneration and atrophy of the centrolobular cells of the liver were always present in some degree; most liver damage was found in the guinea-pig, less in the rabbit, and least in the rat. Intracellular hyaline bodies were found in the liver of the rat alone, and probably represents further development of the same general type of hyaline degeneration as has been observed with certain azobenzenes. Changes in the skin were essentially those of low-grade irritation, and were histologically similar to those of chloracne in man.

Sore Finger-tips in Industry: Protection by Hydrogen Peroxide. BATES, J. V. (1945). *Brit. med. J.*, **2**, 154.

Paper sacks with a rough surface were finished by passing through a wire-stitching machine; this work caused sore finger-tips, and in a period of 6 months 12 women workers reported 192 times to the works surgery. The striking feature about the fingers was the absence of the horny layer of the skin: often drops of blood exuded from the finger-tips. A worker accidentally splashed 20-vol. hydrogen peroxide on her fingers and observed that this prevented the sore. The women now dip their fingers in hydrogen peroxide three times a day, with the result that the same 12 women, in a further period of 6 months, reported only 21 times to the works surgery with sore fingers.

Poisoning by Accidental Drinking of Trichlorethylene. STEPHENS, J. A. (1945). *Brit. med. J.*, **2**, 218.

A man and his wife accidentally took trichlorethylene instead of medicine. The woman, aged 29, complained of spluttering, irritation in her nose, lacrimation, sneezing and vomiting. There was hammering in her head, her legs got weak and she soon passed into unconsciousness, which persisted for 4 hours. She subsequently had abdominal pain, but no jaundice or albuminuria. The man, aged 47, an alcoholic, did not feel any effects for 2 hours, when he became weak in the legs, felt drunk, vomited and passed into unconsciousness. He awoke the next morning feeling 'swimmy' in the head and weak in the legs, and with a dry tongue. He had no other symptoms; no jaundice and no albuminuria.

Acute Paranitraniline Poisoning. MOTASHAW, M. D. (1945). *Indian Phys.*, **4**, 127.

Paranitraniline ($C_6H_4.NO_2.NH_2$) is used in the preparation of red dyes and extensively in the textile industry. It is a yellow powder derived from acetanilide, and is the most poisonous of the aniline group of dyes. Poisoning results from the inhalation of powder or absorption through the unbroken skin. If the powder is ingested on an empty stomach or after alcohol an acute syndrome may result. Four cases of poisoning are recorded. The symptoms included methaemoglobinaemia giving rise to cyanosis and respiratory distress, and a gradually progressive paralysis of the vital bulbar centres which may cause death. Symptoms in the acute condition include progressive weakness, nausea, vomiting and abdominal pain, followed by drowsiness, cold clammy cyanosed skin, and coma. Paranitraniline by its irritative action causes dermatitis, haematuria, cystitis, and carcinoma of bladder, headache, vertigo, and chronic bronchitis. Treatment consists in combating respiratory embarrassment with oxygen therapy, the intravenous injection of methylene blue, together with the administration of stimulants such as leptazol, nikethamide, strychnine or caffeine sodium benzoate.

Industrial Medicine and Respiratory Diseases. WHERRETT, G. J. (1945). *Canad. med. Ass. J.*, **52**, 271.

Industrial medicine is a project in the prevention of disease, and is the first approach to positive adult health. Whereas the death rate from tuberculosis in Canada is 60·4, in unskilled male workers between 15 and 64 years of age it is 184·9, and in semi-skilled workers 102·1. It is thus readily seen that there is a great reservoir of infection to be uncovered and many unknown cases of tuberculosis to be found and treated by modern methods of case-finding. The stimulus to form an industrial medical service can come from the workpeople or management, and where the industry is big enough a full-time service is best. The next best thing is for smaller industries to operate a joint service; and, finally, where this is impossible a part-time service will be necessary. In the matter of rehabilitation it is now generally conceded that it is to the patient's advantage to go back to her old occupation if this is possible, since most people can do work which they know and for which they are trained with less expenditure of energy than something which entails a complete change in their life.

Discussion on Modern Conceptions of Industrial Lung Diseases. SUTHERLAND, C. L., FAWCITT, R., and CRAW, J. (1945). *Proc. roy. Soc. med.*, **38**, 519.

With the exception of silicosis and asbestosis, we are only touching the fringes of knowledge of the pneumoconioses. Abnormal x-ray appearances discovered in workers exposed to dust and fumes do not always signify any present or impending disturbance of health. Fibrosis due to free silica is frequently given the convenient name of classical silicosis, and it is found in such occupations as potter, stonemason and metal-grinder. Pneumoconiosis of coal-miners is defined as 'fibrosis due to silica dust or other dust,' and includes the condition of the lungs known as dust reticulation. It is really an anthraco-silicosis. The collier has become dust-disease-conscious, so that the radiologist in coal-mining areas is being asked to examine an increasing number of chests. Haematite iron ore is a radio-opaque dust, but by itself is innocuous in the lung. Inorganic dust tends to progress from nodulation to coalescent nodulation; whereas organic dust appears frequently to miss the stage of mottling of this type and to progress to bronchiectasis of a massive variety, or, if the cause of infection is removed, the appearance of nodulation may disappear. The x-ray reticulation in miners of this ore shows a very dense radiological shadow, but nodulation without massive fibrosis is a rare condition. Large-sized classical nodulations occur only in the presence of established tuberculosis, and are always associated with solid fibrotic masses. Pathologically, at first there is no stimulation of collagenous fibrosis and practically no increase in the amount of reticulation fibres; later there is tight packing of dust in perivascular locations, with moderate collagen formation.

First-aid treatment of Phosphorus Burns. McCartan, W., and Fecitt, E. (1945). *Brit. med. J.*, **2**, 316.

The facility with which copper combines with active phosphorus to form copper phosphide has been the basis of most methods of first-aid treatment of persons contaminated with phosphorus. Solutions, however, are cumbersome to carry. Four per cent. copper salts in sapo mollis *B.P. Add. III* was, therefore, successfully used. The damage to tissue by burning phosphorus is thought to be due not only to the heat of combustion but also to the intensely hygroscopic action of phosphorus pentoxide, which is the intermediate stage of the change. For this reason a high proportion of water was mixed with the compound. It was also felt that water loosely held in depth over the area by such a compound might act as a better conductor of heat than air. G. R. Cameron and F. Burgess carried out experimental burns and showed that the healing time of wounds was substantially reduced as compared with the standard treatment; and that the substance was much superior to the German $CuSO_4$ paste.

Silicosis in Foundries of Naval Gun Factory. Riley, E. M., Butler, R. W., and Gorem, S. (1945). *Nav. med. Bull., Wash.*, **44**, 653.

Fourteen out of 283 men (4·9 per cent.) employed in dust-hazardous occupations for 20 years or more showed evidence of disease; and 18 men out of the whole foundry of 842 were affected. Foundry dusts contain 20–60 per cent. free silica, and counts of from 5 to 10 million particles per cubic metre are dangerous. The men affected included one ladleman, one crane-driver, eleven moulders and five fettlers. The symptoms were slight, but consisted of cough and shortness of breath. Average dust counts were reduced from 15 million particles per cubic metre in 1939 to 5·2 million particles per cubic metre in 1944 by means of: (1) hydroblast installations; (2) 'Knocking out' outside the foundry; (3) 'knock-out' by means of a pneumatic vibrator; (4) vacuum-cleaning rafters, etc.; by 3 h.p. vacuum cleaner; (5) mechanical sand-mixing; (6) silica and molasses applied by brush instead of spray; and (7) air-supplied abrasive masks complete with heavy duty hoods.

Raynaud's Phenomenon in Riveters. Lindquist, T., and Flemberg, J. (1945). *Acta. med. scand.*, **121**, 309.

A study of the circulatory disturbances in the hands of riveters using compressed-air tools. Four riveters and 6 holders-up, being the men with the most severe symptoms in a group of 150 working in the Gotavarken shipyards, were studied. Their symptoms consisted of a sensation of cold, and stiffness, numbness and blanching of the fingers or terminal phalanges. Damp cold weather was a provoking factor. Measurements of the skin temperature in the finger-tips with a rising body temperature were studied, since this method distinguishes spasmodic and structural disorders in the arteries. In 6 of the men the rise in temperature in the finger-tips was delayed, indicating moderate vascular spasm, but in none was it possible to demonstrate any structural disease. In one other man, however, who had had an accident to his hand followed by suppuration, the temperature did not rise normally, indicating a thrombotic process in the arteries of the two fingers. He was advised to give up all work with tools driven by compressed air.

Paraprol in Industrial Dermatitis. Dobbins, T. (1946). *Industr. Med.*, **15**, 13.

The incidence of industrial dermatitis is greater than any other form of occupational disease, possibly amounting to 65 per cent. of it. Cutting oils may be the most frequent ætiological agent, the workers developing folliculitis and comedones, the hands and hairy portions of the arms and thighs being the areas most commonly involved. Histologically, the process consists of a stimulus to form keratin leading to the formation of a keratogenous plug in the follicular opening from which a folliculitis arises. Paraprol consist of urea, lauryl sulpho acetate and benzyl alcohol in an aromatized demulcent base. Urea is an antiseptic, lauryl sulphoacetate is a detergent having the property of lowering surface tension while benzyl alcohol is a local anæsthetic. Paraprol therefore produces cleanliness, avoidance of irritants and protection of the exposed parts. It was applied in the morning before lunch to be followed by thorough washing, after lunch, and again in the evening to be followed by washing; adequate washing facilities were thus ensured. Three hundred employees exposed to either oils, grease, paints or lacquers reported that a higher degree of protection was afforded than by any other preparation which they had used. The itching was readily allayed and the rash in most instances disappeared in an average of one week. No reactions to the preparation have been observed or reported.

On the Use of Radioactive Tracers in Industrial Medicine. Cantril, S. T. (1946). *Industr, Med.*, **15**, 37.

A radioactive tracer is a small quantity of a particular radioactive isotope which is used to trace and tag a chemical or physiological process. In industry the control of exposure of radium dial painters has utilized radioactive properties in measuring the quantities of contamination on benches, floors and hands, besides measuring the amount deposited in the worker's body by esitmating the radioactive content of the expired air. Since the permissible level of radon content in the air under working conditions is 1×4^{14} curies per c.c., the methods are accurate down to low levels of contamination. Other possible fields of use include the incorporation of a tracer in aniline derivatives; the escape of the tracer from the desired confines in the process or its contamination of working locations or personnel could be measured. At present, 4·5 per cent. of workers with aniline dyes develop papillomata of the bladder by either skin absorption or inhalation. This example could be multiplied in fields such as the manufacture of T.N.T., tetryl, photographic reagents, mercury, lead, arsenic, and the benzols. It must of course be carefully controlled. A radioactive toxin must not be substituted for a non-radioactive one. Once a method is devised for the detection in urine routine urinanalysis on workers would become a further check on the efficacy of the protection programme in force. In looking to the future the use of radioactive tracers may revolutionize the attainment of safe working conditions. The concept of industrial hygiene is prevention of exposure.

Compensable Hernia. Ritter, W. L. (1946). *Industr. Med.*, **15**, 5.

In the United States the compensatibility of hernia is decided by law. In Kentucky compensable hernia must follow an injury. It seems, therefore, the safest industrial policy to prevent those who have a history of hernia in their family, have undescended testicle or other tumour in the inguinal canal, have poor developed musculature, are obese, or have large hydrocœles, from doing heavy work or productive jobs with power driven machinery; where two of the conditions are found in the same individual he should only be allowed to do seated work. Apart from compensatibility the industrial worker is less effective with a hernia. The grounds for restricting employment of those who, on pre-placement examination, are found to have a hernia must rest on the individual's ability to do certain kinds of work with this physical impairment.

Psychosomatic Consultations in Industry. IRVIN, E. A. (1946). *Industr. Med.*, **15**, 1.

'All too frequently physicians view a particular case only from a standpoint of the specialty they practice, and lose sight of the patient as an individual, while others rely almost exclusively on laboratory tests or mechanical aids in making their diagnoses. Often important factors in the emotional side of the patient's life, which may have an effect on his present complaints, are overlooked. Psychosomatic medicine is an essential part of the practice of industrial medicine. To obtain good human relations in an industrial organization, emotional disturbances must be overcome as early as possible, and the earlier the better. Although the emotionally disturbed person is sick, he rarely attempts to see his family doctor, and the chances are that he doesn't even think of consulting a psychiatrist. He just worries through his disturbance or tells his troubles to a sympathetic listener. If there happens to be an understanding factory doctor at his place of employment, he instinctively goes to him. This is an extremely important function of the factory doctor, and it should be recognized as such. The doctor must be aware of the importance of proper job placement for the maladjusted individual. On the other hand it is important not to go so far into psychiatry that some organic disease is missed.

Use and Abuse of Radiographs in the Diagnosis of Mouth Infection. THOMAS, E. H. (1946). *Industr. Med.*, **15**, 9.

Chronic oral infection is an affliction of a great many people and is a direct or indirect cause of many systemic diseases. The bones of the jaws are affected by more abnormal conditions than are bones elsewhere. Numberless acute and chronic conditions are due to the presence of teeth. Teeth sometimes repair themselves after injury, but in most instances are incapable of doing so. They then act as foreign bodies creating continuous irritation to normal tissues over days and years with all of the innumerable pathological conditions resultant therefrom. It is futile to attempt to diagnose disease of the jaw from radiographs without taking a history and making a clinical examination. Radiographs show only changes in calcified bone structure, but many of these are hidden by the shadows of the teeth themselves. At least half of mouth infections occur in the soft tissues and ordinary acute infection such as Vincent's infection show no x-ray changes. On the other hand diseases such as osteomyelitis and neoplastic conditions in the jaw are well shown by radiographs. Clinical mouth examination must include a search for flat contact points, cracked teeth, overhanging fillings, calcareous deposits, ill-fitting dental appliances and tumours. It should also include a check for traumatic occulsion and closed bites as well as an electric test for vitality on all teeth.

THE HEALTH OF MERCHANT SEAMEN.

A CORRECTION.

To measure with any pretence to accuracy the mortality of merchant seamen is notoriously a very difficult task (*vide* the Registrar-General's *Occupational Mortality Supplement* for 1930–32), but in contrasting seamen with males otherwise occupied it is regretted that in a leading article in the issue of this Journal for April 1945 the relative position of the former was, according to the best and latest available figures, made to appear far too unfavourable. The mortality rate of merchant seamen between the ages of 20 and 64 was stated to be ' approximately double the standard rate for the same age group, the death rate from tuberculosis among seamen being four times greater than among the general population.' According to the Registrar-General's analysis the recorded deaths of merchant seamen at ages 20–64 in the 3 years 1930–32 were 47 per cent. in excess of the deaths that would have occurred at the age-mortality rates of all males. In other words, their death rate from all causes at these ages was, 15 years ago, approximately one and a half times and not double the standard rate.

Their excess from tuberculosis (all forms) was almost identical with their excess from all causes of death—namely, 48 per cent. In 1930–32, 1231 such deaths were recorded at ages 20–64, and at the corresponding age rates of all males there would have been 830. Their rate from this cause was, therefore, also one and a half times the rate of the general population of males, and a serious error occurred in referring to it as four times greater. With regard to this disease it was also stated that ' 17 per cent. of all deaths occurring within a year of leaving the sea was due to tuberculosis.' Apart from the great difficulty of arriving at any such figure accurately, it must be noted that no contrast is available for men leaving other occupations to show whether the figure is, in fact, unduly high. It may, however, be observed that for all males aged 20–64, occupied or unoccupied, in 1930–32, as many as $15\frac{1}{2}$ per cent. of the deaths were attributed to tuberculosis.

This leading article arose out of a study by Dr. Jameson Carr in the same issue of health problems in the Merchant Navy. Dr. Carr's discussion of those problems was mainly non-statistical, but where figures do occur the reader should keep in mind the serious difficulties referred to above of measuring the relative mortality of the seaman and in calculating such indices as the proportion of men for whom medical attention is available on board ship.